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JANUARY 1994

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

National advertising representative: State Medical Journal Advertising Bureau, Inc., 11437 West 106th Street, Overland Park, KS 66214. (913) 888-8781

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Official Publication Of the OSMA For 85 Years

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

JANUARY 1994 VOL. 87, NO. 1

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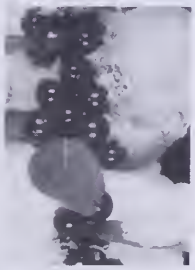

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EDITORIAL

Ray V. McIntyre, MD

Whence Maturity?

Human evolvement to the Promised Land has been glacially slow, intermittent, and attended with acrid argument between the theologic and the anthropologic perspective on evolution. The animalistic body we humans inhabit reiterates the animal substrate. On the other hand, the paucity of residual animal instincts in human function suggests that cognition has largely replaced instinct in humans. Transgenerational education is now required for human racial survival.

But when we see a human baby manage the environment and induce human caretakers to supply what the baby wants, the functional utility of residual instinct is reconfirmed. For the baby, what is wanted is what is needed. We understand the baby is instinctively hungry and "wants" to eat. That the human baby *needs* balanced nutrition, vitamins, electrolytes, warmth, and love is completely outside the expressed "want." The baby has an instinct to eat but not an instinct to eat what is needed. Human ability to separate wants and needs awaits a much more mature stage of development.

The arrival time of maturity is debatable, but a widely used definition of functional maturity states that the mature individual has become able to delay the gratification of instinctive needs to an appropriate time and place (and/or person). Probably the definition of maturity should be enlarged to include the ability to differentiate needs from wants.

Among animals, some needs are filled through an inborn selective appetite that serves to supply what is needed as well as what is wanted. For example, free grazing animals in the wild will migrate many miles to a salt lick for the minerals that herdsmen must supply to captive herds.

An analogous ability in humans is not now recognizable, but it may be that the vestige of an instinctive selective appetite remains present in our human devotion to spicy cuisines. In the Middle Ages, the whole world was explored as human sailors spanned thousands of leagues in search of spices. Today's foods are much flavored before sale. For us humans, spices improve the taste of food, but do not directly improve nutrition. Highly flavored food is now what we want, but not necessarily what we need. Today, human appetite is no longer reliable to select a healthy diet. Learned information must now be used to select a proper human diet.

A wide variety of human troubles results from the propensity to grasp—like a baby—for what we want regardless of what we truly need. Perhaps a more complete definition of maturity will include the idea that the individual has developed an internal ability to recognize the difference between needs and wants. The mature person will satisfy needs in a timely fashion without wasting energy on unnecessary wants.

Ray V. McIntyre, M.D.

G. Lance Miller, MD

"If There's No Risk, It's Not Worth Doing"

Happy New Year!

It has come to be that physicians face each new year with anxiety and dread. We are faced not only with the uncertainty of just what shape and form the new reformed medical system will take, but also with the stress of changes thrust upon us even before the politicians have started their squabbling and compromising.

Before this year is out, your association will offer the physicians of this state an option carefully conceived to make them viable participants in the struggle to control future health care management decisions in Oklahoma.

Your leadership believes the creation of a full service, statewide, physician owned and managed insurance company can and will be a great opportunity for all of us. Opportunity is almost always accompanied by risk, and the cliché "no risk, no gain" certainly applies to this venture.

Last month on television, ESPN featured the ARETE awards presentation for courage in sports. I'm sure many of you watched these awards and were as touched as I by the compelling stories of triumph over adversity.

Many teenagers and young adults were featured, including a high school quarterback with a useless arm; a basketball and tennis star, also with a paralyzed arm; and a young man rendered comatose and paralyzed by an accident, who defied his physician's prognosis and eventually played hockey.

Perhaps most impressive was a gentleman named Hank, who had lost the sight in both eyes to glaucoma. His courageous story of overcoming deep depression was one we all can applaud. This man subsequently took up sailing and sailed across several oceans alone!

During his acceptance speech Hank made what was for me a most telling and insightful observation. He said, "Many people told me not to do it; don't take the risk. If there is no risk, it's not worth doing."

Compared to the adversities of the ARETE nominees, our situation is different but no less challenging. Do we, the physicians of this state, possess the depth of courage shown by the ARETE nominees?

Our risk will not entail the expenditure of supreme mental and physical exertion to overcome organic deficits—only the courage and faith to make a substantial financial commitment. I urge you to examine the importance of your previous investments and compare it to the potential benefits of this venture, both to you personally and to your patients.

"If there is no risk, it's not worth doing."

G Lance Miller, MD

Compression Brachial Plexopathy Caused by Chronic Posterior Dislocation of the Sternoclavicular Joint

Ghazi M. Rayan, MD

Thoracic outlet syndrome developed in a patient due to chronic posterior dislocation of the sternoclavicular joint, following an all-terrain vehicle accident. Decompression of the thoracic outlet was accomplished by surgical reduction of the clavicle, excision of the medial clavicle and reconstruction of the costoclavicular ligament. The patient's symptomatology was relieved by the surgical procedure performed.

THORACIC OUTLET SYNDROME (TOS) IS A compressive disorder of the lower trunk of the brachial plexus and subclavian vessels along the cervico axillary canal. Causative factors include congenital soft tissue anomalies as anomalous fibrous bands, Sibson's fascia, and anomalous insertion of scalene muscles. Congenital osseous anomalies include cervical rib, anomalous 1st rib, long C7 transverse process, bifid clavicle and anomalies of the scapula (Sprengel's deformity) and the spine (Klippel-Feil syndrome). Space occupying lesions, soft tissue or bony such as callus formation following 1st rib fracture, clavicular pseudoarthrosis or malunion are also causative factors.[1-4]

Weakness of shoulder girdle muscles with loss of muscle mass and tone will lead to poor posture and narrowing of the thoracic outlet. This may be triggered by acute trauma. Hypertrophic muscle due to excessive exercise may also lead to narrowing of the space. Repetitive shoulder motion especially with overhead activities (work related or athletic) may lead to traction and compression of neurovascular structures.[4-7]

This article reports a case of TOS caused by chronic retrosternal dislocation of the clavicle.

Case Report

A 44-year-old woman presented with paresthesias and weakness of the right upper extremity and shoulder and upper chest pain of four years' duration. She gave a history of right glenohumeral joint dislocation following a fall from a four-wheeler that landed on her chest. Closed manipulative reduction of the glenohumeral joint was done. The patient had shoulder pain for three years until a chest tomogram was obtained and chronic posterior dislocation of the right sternoclavicular (SC) joint was diagnosed. The patient experienced paresthesias and decreased sensation of the upper extremity and the ulnar two digits. At one time she unknowingly burned her medial forearm with a hot iron. She had progressive weakness and pain of the upper extremity and hand. She also experienced upper medial chest pain, and painful "popping" in the medial clavicle provoked by overhead activities. Sleeping on the right side provoked episodes of nocturnal dyspnea.

On examination the absence of 2-point discrimination in ulnar nerve distribution, including the ulnar two digits, was observed. The 2-point discrimination in the remaining digits was 10 to 12 mm. The patient had weakness of extrinsic and intrinsic muscles of the hand. A hyperabduction test provoked

From the University of Oklahoma Health Sciences Center and Baptist Medical Center, Oklahoma City, Oklahoma

Direct correspondence to Ghazi M Rayan, MD, 3433 NW 56th, #850, Oklahoma City, Oklahoma 73112.

paresthesias, loss of radial pulse, and pain in the medial clavicle. Adson's test did not produce change in the radial pulse. The patient had depression and loss of normal prominence of the clavicle (Fig. 1), and painful limitation and "popping" of the SC joint. The provocative tests for TOS on the contralateral side were normal.

Chest x-rays showed subtle asymmetry of the SC joints. Loss of congruity of the left SC joint and the presence of a bony spike were observed on the tomogram. A CT scan revealed a posteriorly dislocated medial clavicle with malunion of a clavicular fragment (Fig. 2).

At surgery the medial end of the clavicle was found to be displaced in the mediastinum. The medial clavicle, remnants of the meniscus, and fibrocartilaginous tissue were excised. The costoclavicular ligament was reconstructed with a Merseline tape, placed through holes in the clavicular and first rib. The adjacent innominate artery and vein were protected.

The patient's neurologic symptoms began improving three weeks after surgery. At two months she had 7 to 8 mm 2-point discrimination in the ulnar nerve distribution and had painless full range of shoulder motion without instability of the medial clavicle. One year after surgery she was asymptomatic except for occasional discomfort related to glenohumeral joint motion.

Discussion

Thoracic outlet syndrome constitutes a group of disorders including cervical rib syndrome, scalenus anticus syndrome, costoclavicular syndrome, and hyperabduction syndrome. Leffert pointed out that TOS is essentially a clinical diagnosis, and that the most important diagnostic tests are the history and physical examination.[1] Sagging of the shoulders and ptosis of the scapula result from weakness of the shoulder girdle muscles, particularly the trapezius, levator scapulae, and rhomboids. This leads to traction on the inverted U-shaped lower trunk of the brachial plexus over the first rib and narrowing of the costoclavicular space, i.e., costoclavicular syndrome. This postural abnormality is a common etiologic factor of TOS. Rehabilitation and strengthening of the shoulder girdle muscles is recommended for these patients.[6,8]

Thoracic outlet syndrome may be associated with sternoclavicular hyperostosis, a condition characterized by progressive painful enlargement of the sternum, clavicles, and rib cartilage. Indomethacin is believed to control these patients' inflammatory symptoms.[9] Surgical treatment of TOS is indicated in the presence of severe neurologic or ischemic symptoms and following a period of nonoperative treatment without favorable response. The choice of surgery is dictated by the pathology. Surgical decompression of TOS can be accomplished by scalenotomy, scalenectomy, claviculectomy, pectoralis minor release, first rib resection, and cervical rib resection.[1] Transaxillary first thoracic rib resection is the most commonly used procedure for decompressing the contents of the thoracic outlet.[2,10]

Posterior dislocation of the SC joint is rare and associated with more serious complications than anterior dislocation because of the vital structures in-

Figure 1. Normal clavicular prominence can be seen on the left side, and loss of clavicular prominence can be seen on the right.

Figure 2. CT scan at the level of the sternoclavicular joint shows normal left medial clavicle and distortion with posterior displacement of the clavicle along with a bony fragment on the right side.

volved.[11] Of 60 SC joint dislocations reported by Nettles and Linscheid, only 3 were retrosternal.[12] The SC joint ligaments include the intraarticular disc ligament, costoclavicular ligaments (anterior and posterior), capsular ligaments (sternoclavicular anterior and posterior), and interclavicular ligament.[11] Following SC joint dislocation these ligaments are disrupted. The mechanism of retrosternal dislocation of the SC joint is either direct force applied to the anterior clavicle or indirect force applied to posterolateral aspect of the shoulder.

Gangahar and Flogaites[13] reported a case of TOS and retrosternal dislocation of the clavicle. Their patient was recognized initially to have sternoclavicular dislocation and the symptoms were more vascular than neurological. In this case the diagnosis of posterior dislocation of the SC joint was missed for three years after the initial injury, and the patient's symptoms were predominantly neurologic. Decompression of the neurovascular structures in this case of TOS due to chronic posterior SC dislocation was achieved by reduction of the dislocated clavicle, excision of the medial clavicle, and reconstruction of the costoclavicular ligament.

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The Author

Ghazi M. Rayan, MD, is clinical professor of orthopedic surgery at the University of Oklahoma Health Sciences Center in Oklahoma City.

Mohs' Micrographic Surgery and Prompt Reconstruction for Basal Cell Carcinoma: Report of 62 Cases Using the Combined Method

William Sahl, MD; Randall Yessenow, MD; Juan Brou, MD; Norman Levine, MD

Aggressive, recurrent, and difficult to manage skin cancers may require complex solutions. In this review of 62 skin cancers, we demonstrate a novel approach to the management of these tumors. Mohs' micrographic surgery was used to remove the tumor, and was followed by immediate or delayed plastic surgical repair. Using this combined method, the highest attainable cure rate is linked with a coordinated prompt cosmetic repair.

In this series of 54 patients with 62 skin cancers, there was one recurrence with a follow-up of one to four years. Most cases (65%) involved reconstruction within ten days of tumor removal. Eighty-one percent had a single-stage reconstruction. In seventeen patients, new tumors developed at other sites during the four-year follow-up period.

On the basis of our experience with this combined method, we feel that Mohs' surgery, coordinated with prompt plastic repair, offers distinct advantages in the management of difficult skin cancers.

History

CUTANEOUS MALIGNANCIES REMAIN A CHALlenge to all of those involved in their treatment. More than 500,000 estimated new cases of skin cancer are reported each year.[1-3] Basal cell carcinoma accounts for the majority of these lesions. Approximately 90% of these patients are cured by the initial therapeutic modality employed.[4,5] The primary techniques used include simple excision, electrodesiccation and curettage, cryotherapy, and irradiation. It is significant to note, however, that between 10,000 and 15,000 patients present with recurrent tumor each year.[4] Mohs' surgery has been found to be useful in the management of these recurrent lesions as well as in selected primary lesions. In addition to its reliability, Mohs' surgery allows for the maximum conservation of normal tissue.[6,7] The plastic and reconstructive surgeon assumes a prominent role in the reconstructive effort following Mohs' surgery.[8]

In 1941, Frederic Mohs presented his initial results using micrographic surgery.[9,10] His technique at that time involved the application of zinc chloride fixative. This was followed by excision of the cutaneous malignancy using frozen section microscopic control. The fixative was formed by a combination of zinc chloride with granulated stibnite, and an extract of sanguinaria canadensis.

The product of these ingredients is a thick, black paste.[11] The paste is actually a caustic, escharotic fixative agent that allows for the excision of lesions in a relatively bloodless field. It also preserves the tissues so that they are not distorted in microscopic sections.[9,10,12] Many authors have described their personal modification of the fixed tissue techniques. However, the basic principles and techniques are as follows: (1) debulking the tumor, (2) application of the zinc chloride paste which is left intact for 12 to 24 hours, (3) excision of a layer of fixed tissue, (4) mapping, cutting, and staining of the margins, (5) examining further horizontal sections to determine any areas of persistent tumor, (6) repeating steps 2-5 until

From the Department of Dermatology and Section of Plastic Surgery, University of Oklahoma Health Sciences Center, Oklahoma City

Direct correspondence to Norman S. Levine, MD, Professor and Chief, Section of Plastic Surgery, P.O. Box 26901, Oklahoma City, OK 73190

no residual tumor is present, and (7) allowing the wound to heal by secondary intention.

The published reports using this technique have been quite encouraging. Mohs' surgery has been shown to result in 99% five-year cure rates for primary basal cell carcinoma[13-15] and 94% cure rates for recurrent basal cell carcinomas.[9,15-17] In the treatment of squamous cell carcinomas, Mohs reported a 94% five-year cure rate for primary lesions, and an 84% cure rate for recurrent lesions. [13]

One of the major modifications of the original Mohs' technique involved elimination of the fixative. This subsequently became known as the fresh tissue technique. Tromovitch and Stegman published the first long-term follow-up using the fresh tissue technique in treating basal cell carcinomas.[18] The results using the fresh tissue technique were quite comparable to the fixed tissue technique. It soon became apparent that there were several other advantages to the fresh tissue technique. First was the fact that the perioperative pain from in situ tissue fixation was eliminated.[19] Also, multiple stages of the procedure could be performed in a single day. It was found to be useful in most sites, including the periorbital area. Less post-operative bleeding for the surgical site was also noted.[12] Finally, the post fixation tissue slough was eliminated. Accordingly this allowed for the possibility of immediate reconstruction of the defect.[11] For the most part, most Mohs' practitioners currently employ the fresh tissue technique.

Technique

Fresh tissue Mohs' micrographic surgery is best performed in an outpatient setting using xylocaine local anesthesia. A general evaluation of the clinical extent of the tumor is made and adequate anesthesia is administered.

The first layer is made using a saucerized excision with a scalpel (Fig. 1A-F). The purpose of the first layer is two-fold. First, all obvious tumor is removed and second, pathological evidence of tumor type and biological activity can be provided.

The second and subsequent layers are obtained in much the same manner. A saucerized portion of tissue is obtained which includes a margin of normal skin and all the tissue from the wound base. Ordinarily the tissue obtained is 1 to 3 mm in thickness and represents the totality of tissue subadjacent to layer number one.

The pallet of tissue representing layer number two is divided into sections, and these sections are numbered, colored with dyes, labeled, and mapped. Each of these sections is mounted in a cryostat and cut to provide the entire under surface of the section of the slide. Staining with hematoxylin and eosin is done in the usual manner. In this way, the tissue is examined section by section to provide total surveillance of the wound. The Mohs' operator marks the map corresponding to the site of remaining tumor and further excision of the tumor at the wound site is made. This procedure is carried out until the wound site is clear of residual tumor.

With this technique, perineural tumor can be followed microscopically, and bone, cartilage, muscle, and all soft tissue can be removed with the greatest conservation of uninvolved tissue.

Materials and Methods

Between January 1, 1988, and December 31, 1991, 54 patients underwent Mohs' micrographic surgery for 62 lesions by our dermatologic service and were subsequently referred to the plastic surgical service for reconstruction. In most cases, the referral was made immediately following Mohs' surgery if the defect was large enough to require it. In some cases, either because of difficulty of margin resection or because of extreme multicentricity of the lesion, referral for reconstruction was not made for at least one year and sometimes longer following tumor resection.

The tumors were nearly all of the head and neck, and their specific locations are summarized in Table 1.

Results

The 54 patients studied range from 30 to 86 years of age. Twenty-nine patients (54%) were male and 25 patients (46%) were female.

In most patients (65%), reconstruction was performed within 10 days of ablation, and in 13 (24%) it was done on the same day (Table 2). Reconstruction on 10 patients was performed between 11 and 30 days following ablation. Such a delay was generally occasioned either by patient convenience or by unfavorable wound conditions requiring dressing changes at home. In 4 patients (8%) reconstruction was done from one month to one year following surgery. Five patients (9%) waited between one and four years for reconstruction. This latter group usually included those patients in whom a delay was thought advisable because of the multicentricity of the skin cancer.

Reconstruction in all but 10 patients was done in one stage (Table 3). In 17 of these patients only a skin graft (full thickness or split thickness) was required. One stage repair, either a flap or a combination of flap

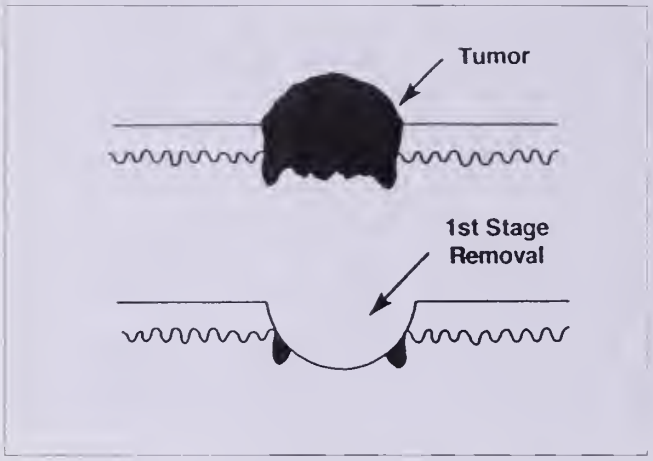

Figure 1A. Cross sections of tumor mass and representation of debulking removal.

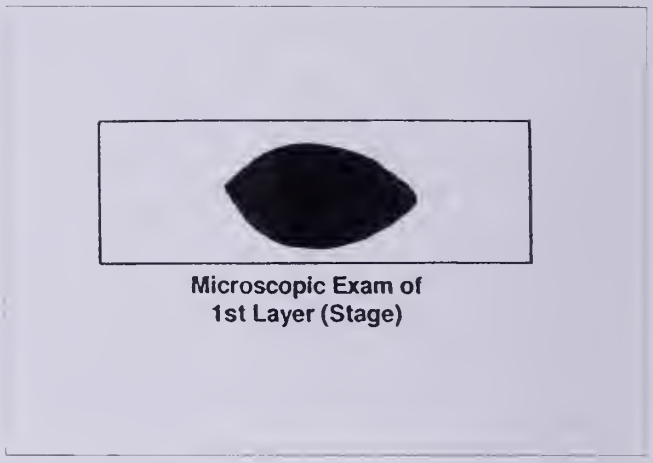

Figure 1B. Gross tumor in first layer for diagnosis.

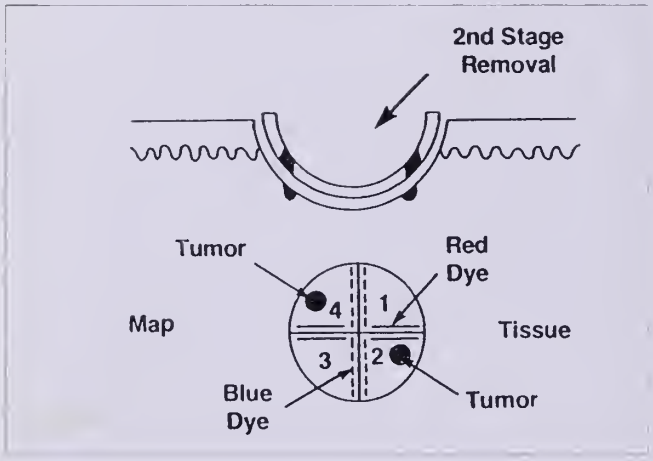

Figure 1C. Removal of next layer of tumor and Mohs' map showing residual tumor at base.

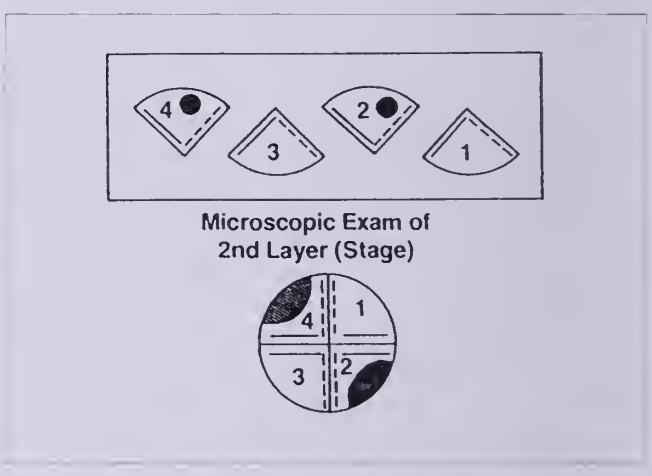

Figure 1D. Microscopic examination of second stage indicating presence of tumor and Mohs' map demonstrating the location.

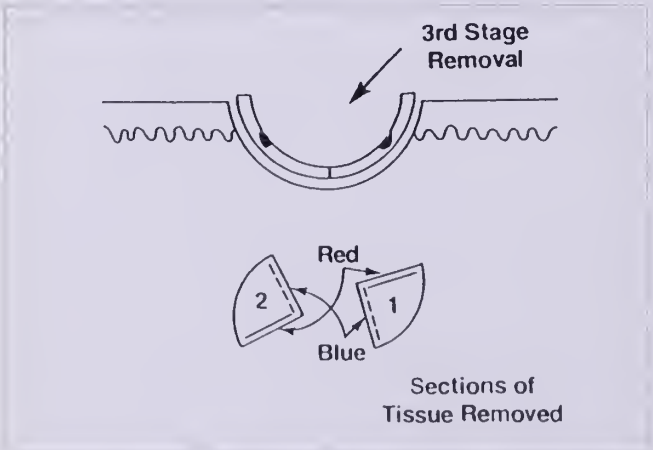

Figure 1E. Third stage removal of only the two involved sites.

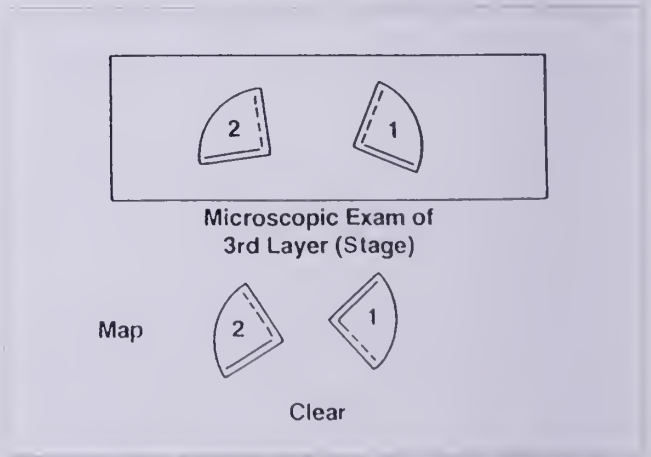

Figure 1F. Microscopic examination showing no residual tumor and Mohs' map indicating the site is clear of tumor.

and skin grafting, was done in the 27 remaining patients. Ten patients had multiple staged operations for lesions of either the nose or the ear. These required composite reconstruction of the associated cartilaginous or bony framework and, in the case of the nose, the nasal lining. From two to five procedures were required to reconstruct these patients.

Two complications occurred in our patients. Both were in elderly patients undergoing multistaged nasal reconstructions. These involved partial flap loss (2 patients) and the associated exposure of a nasal bone graft in 1 patient.

Despite the fact that our patients were selected because of the tendency of the tumors to recur, we had only one patient with recurrence at the site of the lesion. This patient was a 70-year-old woman whose reconstruction was delayed for three years because of the multicentricity of the tumor at the primary resection. She developed a small recurrence at the suture line of nasal reconstruction, which was corrected with excision and advancement of the surrounding tissue. This low recurrence rate for a one-to-four-year follow-up is particularly significant in view of the fact that in 9 patients (17%) new primaries developed in sites other than that of the initial resection.

Discussion

The effectiveness of Mohs' surgery in the treatment of cutaneous malignancies has already been mentioned. The question is which lesions should be excised using Mohs' surgery. Recurrent basal cell or squamous cell carcinoma accounts for the largest number of procedures.[20] Basal cell carcinoma is the most frequently occurring cancer in human beings, with more than 80% of these being present on the head and neck.[11] Most primary lesions can be treated by the routine techniques already mentioned.

Primary cutaneous malignancies for which Mohs' surgery is indicated include[12]: (1) lesions with a high recurrence rate; (2) tumors with poorly delineated clinical borders; (3) large lesions (>2 cm); (4) aggressive and infiltrative lesions; (5) lesions with aggressive histologic subtypes—morpheaform or sclerotic, multicentric, infiltrating, or baso-squamous types of basal cell carcinoma; (6) areas which necessitate maximal tissue conservation, e.g., eyelid, nose, finger, penis; and (7) tumors expressing perineural invasion.

Mohs' surgery is also indicated for other lesions. Incompletely excised basal cell carcinoma is an accepted indication. Although some physicians are content to watch these cases closely, other studies have shown this to be a somewhat dangerous practice. Pasal[21] showed that when tumor was present to within one high-power field (400∞) of a surgical margin, a 12% recurrence rate was present. If the tumor directly involved the margin, there was a 33% recurrence rate.

Mohs' surgery is the treatment of choice for recurrent basal cell carcinoma.[15] Mohs' surgery has shown a success rate of 94% for these lesions, compared to only 80% when treated by traditional methods.[15]

We feel that patients appreciate and respect the expertise provided by both the Mohs' surgeon and the plastic surgeon. The ablation is performed by one physician with the most reliable means available to ensure adequate tumor removal. Likewise, the reconstruction is performed by a physician well versed in this field. This type of procedure and protocol also eliminates any bias on the part of the plastic surgeon if he or she also had performed the ablation. It prevents the reconstructive surgeon from compromising the surgical margins and unduly trying to avoid certain structures (e.g. lacrimal apparatus) by thinking of the reconstruction at the same time. The plastic

Table 1. Location of 62 Skin Cancers

Area	Number	Percent
Nose	28	44%
Cheek	9	14%
Ear	8	13%
Eyelid	5	8%
Lip	3	5%
Leg	1	2%

Table 2. Interval Between Tumor Removal and Reconstruction

Interval	Number	Percent
Same day	13	24%
1-10 days	22	41%
11-30 days	10	19%
1 month-1 year	4	7%
1-4 years	5	9%

Table 3. Follow-Up (54 Patients, 1-4 Years)

Type of Case	Number	Percent
Single stage reconstruction	44	81%
Multiple stage reconstruction	2	4%
Complications (wound)	2	4%
Recurrence	1	2%
New primaries	9	17%

surgeon is presented with the excisional defect and must then provide adequate reconstruction.

By using the fresh tissue technique, the possibility of immediate reconstruction is made possible. Patients are able to visualize and adjust to the defect created by the Mohs' surgeon. Those patients don't necessarily require reconstruction on the same day as excision. However, some patients have a great deal of difficulty dealing with the defects created. If the proper criteria have been met, these patients have been greatly relieved and are appreciative of immediate reconstruction. Several of the patients in this study were involved in occupations requiring a great deal of personal contact. Immediate reconstruction allowed them to return to their jobs much more quickly. Some examples of lesions for which patients have requested immediate reconstruction include: composite nasal alar defects, medial canthal defects, and lower lip defects.

There are also several functional indications for immediate reconstruction. Lesions involving the eyelids or medial canthal areas should be repaired soon after Mohs' surgery to prevent ectropion and the associated complications of corneal exposure.[22] Large defects of the lip require immediate reconstruction to prevent drooling and the associated problems of oral incontinence.[11] Another example is that of a scalp defect extending to the level of the dura mater of the brain. Closure is essential for protection of the underlying brain.[8]

There are several other advantages of using Mohs' micrographic surgery that should be addressed. As alluded to earlier, the technique described is extremely accurate and reliable. Standard vertical sectioning of specimens has been shown not to be as accurate as horizontal sectioning in Mohs' surgery.[23,24] This is due to the fact that neoplasms do not grow symmetrically. Rather, tumors extend unpredictably due to resistance of nearby structures.[12] Tumors have also been shown to extend along embryologic fusion planes. This is most dramatic with both primary and recurrent lesions in the head and neck.[25] Mohs' surgery also allows for the complete excision of the tumor with the conservation of the maximal amount of normal tissue.[12] Instead of randomly selecting a surgical margin, the entire under surface and all peripheral margins are examined. The technique also allows for elimination of the costly operating room expense while waiting for the report of frozen section margins. Finally, reconstruction following Mohs' surgery can be undertaken in an ambulatory surgical setting. Almost all post Mohs' reconstruction can be done using local anesthetic.

There also are disadvantages to the combination of Mohs' surgery and reconstruction. It involves two separate procedures with the associated costs of both. However, the accuracy and reliability of the microscopically controlled excision is justified in spite of the possible inconvenience. The added cost of waiting for frozen sections in the operating room also should be balanced against the outpatient cost of Mohs' surgery.

It is obvious that Mohs' surgery with immediate reconstruction should not be used for every cutaneous malignancy. More specifically, there are several instances where Mohs' surgery may be the recommended treatment for excision, but reconstruction should be delayed. Lesions which fit into this category include neoplasms which have recurred multiple times, extremely large lesions, lesions occurring in areas which have previously been irradiated, and lesions occurring in areas where the potential for recurrence is great even following Mohs' surgery (e.g. lesions in the area of embryological fusion plane).[11] Management of these patients should include delayed reconstruction. An alternative is to place a thin split thickness skin graft over the defect.[11,26,27] If there is no evidence of recurrence in 12 to 18 months, further reconstruction may be attempted.

Finally, one of our most recent cases demonstrated an instance where Mohs' surgery can be somewhat limited. A recurrent infra-auricular lesion was initially excised by Mohs' surgery. However, when we attempted to clear the deep margin, we found tumor was still present at the level of the superficial lobe of the parotid gland. At this point, the patient was referred to the plastic surgeon for further excision to clear the deep margin, with attempted preservation of the facial nerve branches. A superficial parotidectomy in the operating room was performed. We believe that when tumors extend into the parotid gland, consideration should be given to tumor excision in the surgical suite to allow identification and dissection of the facial nerve. Continued horizontal sectioning of lesion by the Mohs' surgeon at the level of the parotid gland might cause undue risk to the facial nerve, in certain cases. This is a relative contraindication, and is the only instance we have encountered where the Mohs' technique might have a disadvantage.

Conclusion

Skin cancers with a propensity to recur, because of either previous recurrence, cell type, or multicentricity, represent a challenge to anyone involved in their treatment. We believe that a two-team approach offers some real advantages to these patients. Ablation is performed by the Mohs' micrographic surgeon, who is well oriented to the sections that he subsequently reads. The patient is then referred to plastic surgery for reconstruction. When embarking on this study, we were concerned about the convenience to the patient of requiring two separate operative procedures. However, of the 54 patients on whom resections were performed, 35 (65%) underwent reconstruction within 10 days of cancer removal. Thirteen patients (24%) underwent their reconstruction done on the same day.

Although two operative procedures were required, the majority of both the ablations and reconstructions could be done under local anesthesia. Long delays in the operating room waiting for frozen sections were avoided. The costs of operative time and multiple frozen section examinations taken perpendicular to the lines of resection were avoided.

The benefits of this approach include certainty of margins, planar sectioning of the histologic specimens, tumor ablations that are performed without concern for reconstruction, and the avoidance of long operative procedures and multiple frozen section examinations. We feel that these advantages outweigh the disadvantages cited above. We have tried to combine the expertise of two disciplines for the patients' benefit and convenience.

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The Authors

William J. Sahl, MD, was formerly associate professor of dermatology and chief of dermatologic surgery at the University of Oklahoma Health Sciences Center. Currently he is in private practice in Rapid City, South Dakota.

Randall Yessenow, MD, completed his residency training at the University of Oklahoma in 1991. He is currently in private practice in Merrillville, Indiana.

Juan A. Brou, MD, is assistant professor of surgery at the University of Oklahoma College of Medicine.

Norman S. Levine, MD, is professor and chief, Section of Plastic Surgery, University of Oklahoma.

Percutaneous Discectomy

Douglas L. Polk, MD

The present study represents a retrospective evaluation of 110 patients with symptomatic lumbar disc disease treated by percutaneous discectomy. In the group of patients without prior lumbar surgical treatment or pending workman's compensation (Group 1), the outcome from surgery was good or excellent in 75%. The patients that represented workman's compensation/liability cases (Group 2), or had a history of prior surgery (Group 3), had good/excellent results in about 50% and 0%, respectively. In all groups, there was no morbidity or mortality. The benefits of percutaneous discectomy are discussed, and include shorter hospitalization and less cost to the patient. As compared to the more conventional (open) spine procedures (lumbar laminotomy and discectomy), the automated or newer laser discectomy appears to be a reasonable alternative in a large number of patients, except those who have had prior lumbar spine surgery.

LOW BACK PAIN ORIGINATING FROM PATHologic lumbar intervertebral discs is one of the most common disorders afflicting human beings. An estimated 80% of adults will experience one or more episodes of low back pain in the course of their lifetime. An estimated 350,000 surgical procedures are performed on the lumbar spine annually in the United States alone.[1]

There have been many recent advances in evaluation and management of lumbar spine disorders, including diagnostic techniques such as MRI and new surgical innovations. There are a number of different surgical procedures for treatment of lumbar spine conditions, tending to confuse the lay public as well as physicians as to the proper utilization in a particular patient.

Of the many procedures in use, the most commonly performed spine operation is the standard lumbar laminectomy and discectomy (which is actually a partial nuclectomy; the entire disc is not removed). Numerous variations of this procedure exist today, including lumbar microdiscectomy. These procedures can be combined with bony fusion and with or without various types of instrumentation (internal fixation). The recent reintroduction of the interbody fusion can be done either anteriorly or posteriorly. Another less invasive, probably less effective and yet still accepted treatment modality also includes the intradiscal injection therapies (chymopapain or collegenase). Thus, it is apparent that the treatment of the lumbar spine encompasses a wide variety of treatment modalities, surgical and non-surgical.

Chymopapain nucleolysis initially met with a great deal of enthusiasm, as usually occurs with new treatment modalities. The long-term results were ultimately somewhat disappointing and a number of spine surgeons, including the author, have discontinued its use. Failure of chymopapain nucleolysis to relieve symptomatology has been reported as high as 50% in some series. More recently, when Onick and his colleagues introduced automated percutaneous discectomy as a new, relatively less invasive technique, it was viewed with a degree of skepticism.

From the Oklahoma Neurological Surgery Clinic, Inc., 4140 W. Memorial Road, Suite 300, Oklahoma City, OK 73020.

Direct correspondence to Douglas L. Polk, MD, 4140 W. Memorial Road, Suite 300, Oklahoma City, OK 73020.

However, the relative failure of chymopapain as an effective treatment option for lumbar disc syndrome prompted the evolution of a newer form of percutaneous discectomy, in which a cannula is placed, under local anesthesia, into the proper location in the intervertebral disc space with the aid of fluoroscopy. The disc material is then separated by a reciprocating cutting blade which is placed through the cannula, and then this separated disc material is removed by suction.

Material and Methods

The study groups consisted of 110 patients* who underwent percutaneous discectomy. Patients were selected for surgery based on standard clinical criteria consisting of a painful lumbar disc syndrome with confirmatory physical findings; positive straight leg raising, neurological deficits, etc., and refractory to at least three months of conservative therapy. Radiographic tests (CT, MRI, and myelography, or a combination of these) confirmed the presence of one or more prolapsed (or herniated) discs that were anatomically appropriate for the patient's symptoms and signs. Of the 110 patients who underwent this procedure, we were able to follow-up with 81 (74%). The follow-up consisted of direct interviews of patients by medical personnel; telephone interviews; and by mailed questionnaires.

In order to measure the relative success of the procedure, in regard to pain relief and function, there were a number of different parameters used to rank the patient with the most significance given to the subjective view of the patient. Other factors considered were time after surgery that the patient returned to work, functional activity levels, use of pain medication, etc. The follow-up interviews ranged from 2 months to 12 months after the surgery, with an average follow-up time of 3.8 months.

The outcome was defined as follows:

Excellent: Full working capacity; pain free or only very minimal pain requiring no medication.

Good: Full working capacity; mild, residual low back or radicular pain, sometimes requiring mild analgesics.

Fair: Reduced working capacity; continued low back pain and/or radicular complaints. This group was "minimally improved" from what the patients were preoperatively, but they were *definitely* better than they were before the surgery.

Poor: Reduced work capacity; requiring analgesics. This group was essentially no better than the patients were before surgery, and the operation was considered "unsuccessful."

The patients were divided into three groups based on their histories. The first and largest group of 39 patients (Group 1), we call the "standard" group. These patients had no particular or unusual historical features. The second group (Group 2), consisting of 34 patients, represented workers' compensation claimants or other types of liability cases. The eight patients in the third group (Group 3) were patients who had undergone prior lumbar spine surgery.

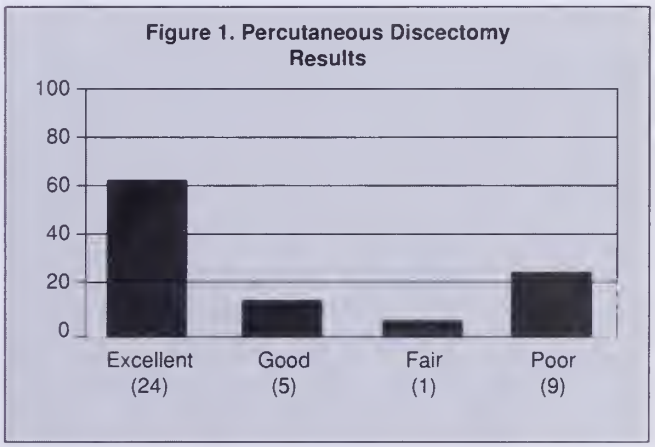
Results

As indicated in Figure 1, the standard group of patients (Group 1) did very well following percutaneous discectomy. Excellent results were reported by 62% of the patients in this group. Good results were reported by 13%, fair by 3%, and poor by 23%. Thus, 75% of the 39 patients in this group reported good or excellent results.

The workers' compensation group (Group 2) clearly did not fare as well (Fig. 2). Excellent results were reported by only 9% of this group. Good results were reported by 35%, fair by 3%, and poor by 53%. Thus, only 44% of the 34 patients in this group reported good or excellent results.

The group of patients who had prior operative procedures (re-do percutaneous discectomy or prior open lumbar spine surgery) (Group 3) did not do as well. There were no patients in this group that reported either good or excellent results (Fig. 3). Fair results were reported by 50% of the patients, poor results by the remaining 50%.

The majority of these operations were done on an outpatient basis. A small percentage had a short hos-



*Treating physicians were A.C. Lisle, Jr., MD; B.J. Rutledge, MD; D.F. Rhinehart, MD; Stan Pelofsky, MD; G.W. Schoenhals, MD; Stephen K. Cagle, MD; Douglas L. Polk, MD; and Robert L. Remondino, MD.

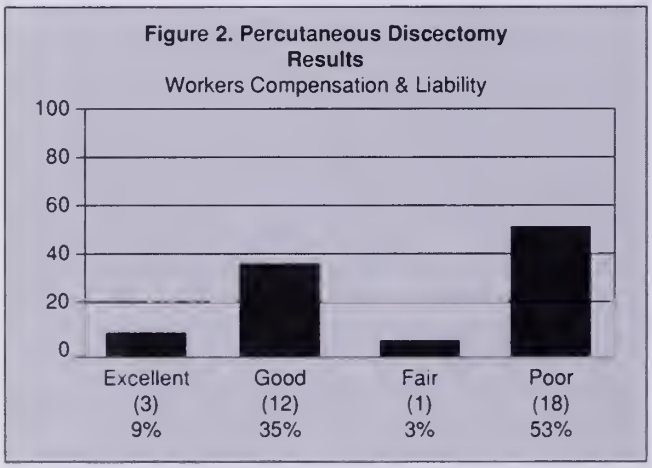
pital stay, generally one day. There were no operative complications. There were no infections or neurologic complications. In no case were any patients considered to be "worse" following the surgery.

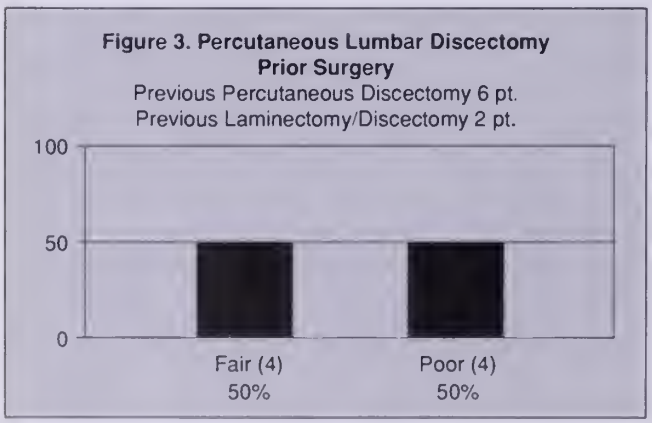
Of the patients that were treatment "failures" with percutaneous discectomy, a number went on to have an open spine procedure, most commonly a lumbar microdiscectomy, sometimes combined with fusion and only rarely with fusion and instrumentation.

While we do not have precise follow-up on these (failed percutaneous discectomy) cases, the majority of patients that underwent a second procedure (usually an open lumbar microdiscectomy) were improved and were satisfied with their results.

Discussion

It is evident from these results that percutaneous discectomy offers a reasonable alternative to open lumbar spine surgery for a selected group of patients. The 75% rate of significant improvement or "successful" surgery reported by the standard group (Group 1) compares very favorably with other series using the standard open operative procedure (lumbar laminotomy and discectomy).





Regardless of the procedure used, patient selection is of paramount importance. The optimum candidates for this procedure are patients under 50 years of age with a single level herniated nucleus pulposus contained within the disc space. Those patients with disc fragments not contained within the disc space (free intraspinal fragments) or those with bony compression (lumbar stenosis) would not be candidates for this percutaneous procedure. Patients who have predominantly radicular (leg) pain as opposed to low back pain also tend to have superior results as compared to those with a less poorly defined radicular component.

Percutaneous discectomy offers several advantages over open surgical techniques. The most important advantage is less postoperative discomfort, with reduced tissue destruction, leading to a faster recovery. Moreover, because the spinal canal is never entered, there is less chance for epidural scar tissue or epidural fibrosis (which is one of the well-known causes of "failed back syndrome").

Based on national studies, the percutaneous discectomy is clearly safer than the open procedure, with a morbidity and complication rate approaching zero.[2]

Finally, this procedure is generally done on an outpatient basis, making it cost effective, and in line with the current trend toward containment of health care expenditures.

The chief disadvantage of the procedure is that it is not effective when there is any significant component of bony stenosis, ligamentous hypertrophy, or other foraminal encroachment on the neural elements. Therefore, it excludes most elderly patients, and it certainly would not be applicable to patients with a "free fragment" or "sequestered fragment" of discs. Also, the L5 S1 level is often difficult to treat with this procedure.

Our latest approach to percutaneous lumbar discectomy has been with the use of a Holmium YAG laser technique. Our experience is still too limited to compare quantitatively to percutaneous automated discectomy technique described in this paper.

Summary

1. In patients with symptomatic lumbar disc herniations contained within the interspace, the percutaneous automated discectomy is a reasonable option to open operative procedures. The results appear to be comparable in either case.

2. The procedure is done under local anesthesia,

thus avoiding a general anesthetic, and has a shorter and less painful postoperative recovery period.

3. The complications and morbidity from the percutaneous technique appear to be significantly less than the open surgical techniques.

4. Proper selection of surgical candidates is extremely important in any surgical approach to the treatment of lumbar degenerative disc disease. Patients who have had a prior lumbar surgery (or "redo" cases) do not appear to do well with percutaneous discectomy.

5. As demonstrated in many other studies, the workers' compensation/liability patients did not fare as well as "standard" patients.

6. Since the original study, we have performed an additional 50 automated percutaneous discectomies and 52 laser discectomies (totaling 212 cases in all).

7. Although the sample size is small, and our experience is limited, patients treated with laser discectomy appear to be experiencing a higher percentage of favorable results than patients treated with automated percutaneous discectomy.

Acknowledgments

The author would like to thank Teresa Bradshaw, RN; Michael Pollay, MD; Don F. Rhinehart, MD; Sandy Felder; and Mr J E Reynolds for their assistance in preparation of the manuscript.

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The Author

Douglas L. Polk, MD, is currently a clinical assistant professor at the University of Oklahoma College of Medicine and has a full-time private practice in neurological surgery in Oklahoma City.

Jungle Medicine

An Oklahoma Medical Team Visits Mexico

R. Richard Edde, MD; Sandi White, RN, MS

FIVE HUNDRED EAGER PATIENTS GREETED the arrival of our bus in the remote jungle village of Kan-tunilkin, Mexico. Located in the Yucatan peninsula and a two-hour journey north and west of Cancun, this isolated Mayan community was the destination of our 15-member medical team from Oklahoma during the last week of January 1993.

We had come to Kantunilkin to provide much-needed medical care to a people whose way of life is directly affected by no sanitation, non-existent safe drinking water, no refrigeration, and a dependency on farming and hard physical labor.

We immediately set to work organizing a thatched hut into a pharmacy stocked with medicines we had purchased in the U.S. and brought with us. Within an hour, we were seeing patients in make-shift examination cubicles put together with rope and blankets.

The Oklahoma medical team consisted of triage nurses Cathy Edde, RN, and Sandi White, RN; with Marcilino Lopez, RN, Karen Smit, RN; gynecologist Roger D. Quinn, MD, and his wife Joy Quinn, RN, who is a gynecology nurse clinician; R. Richard Edde, MD; Amir Soas, MD; Joseph E. Leonard, MD, and his wife Clinette; tax attorney Jonathan Reiff, along with his daughter Sherry; and project coordinator Reverend L. Dale DePue rounding out the rest of the team.

Intestinal parasites; anemia; gynecological complaints; pregnant women; children with ear, nose, and throat problems; and generalized aches, pains, and strains accounted for the majority of the patients seen. Long lines were inevitable as people traveled from all over the peninsula in hope of a cure. We encountered several patients who needed more extensive evaluations than available in the village, and through a Mexican government social worker arranged further care for them in Cancun. Table 1 shows the number of patients in each diagnostic category.

Patients wait to be seen by the clinic's medical team.

A young woman near death was delivered to the clinic one morning. She had a draining intra-abdominal abscess and was extremely dehydrated. Life-saving supportive care was rendered this patient by our doctors, and she was transferred to Cancun. She most likely would have died in the absence of any medical care.

Direct correspondence to R. Richard Edde, MD, 6920 Lakepoint Drive, Oklahoma City, OK 73116

A mother brought her 18-month-old child to the clinic because of a swelling on the child's back. The diagnosis was spina bifida, and upon returning home, several members of the medical team began arrangements to bring child and mother to the States for definitive surgery.

Table 1. Numbers of Patients by Diagnosis (Patients May Have Multiple Diagnoses)

Intestinal parasites	177
URI/Tonsillitis	82
Muscle strain/aches & pains	32
Skin disorders (fungal & allergic)	34
Arthritis	37
Urinary tract infection	25
Anemia	25
Gastritis/reflux	16
Eye complaints (conjunctivitis, pterygium, cataracts)	14
Pregnancy	13
Allergic rhinitis	10
Bronchitis	10
Menstrual complaints	10
Vaginitis	9
External otitis	8
Pelvic inflammatory disease	7
Malnutrition	7
Diabetes	6
Cholecystitus or gallbladder symptoms	6
Headache	5
Hearing loss	5
Perforated tympanic membrane	5
Cerumen impaction	5
Diarrhea	5
Miscellaneous:	
Sinusitis	2
Seizure disorder	3
Urethritis	1
CA liver	1
Uterine fibroids	2
Prostatitis	3
Shingles	1
Infertility	3
Hypertension	3
Nasal septal perforation	1
Tonsillar mass	1
Costochondritis	2
Abnormal heart murmur	2
Wounds	3
Facial nerve paralysis	1
Urethral stricture	1
Inguinal hernia	1
TMJ	4
COPD	2
Serous otitis media	4
Dizziness	1
Endometritis	1
Intrauterine fetal death	1
Epistaxis	2
Atrial fibrillation	1
Lipoma	1
Abdominal abcess with sepsis	1

Approximately $5,000 in medicines were dispensed, the money donated by the First Presbyterian Church in Edmond, Oklahoma, and physicians of Central Oklahoma Anesthesiology.

Table 2 shows the list of medicines used in Kantunilkin. Supplies were donated by Presbyterian Hospital in Oklahoma City, Oklahoma.

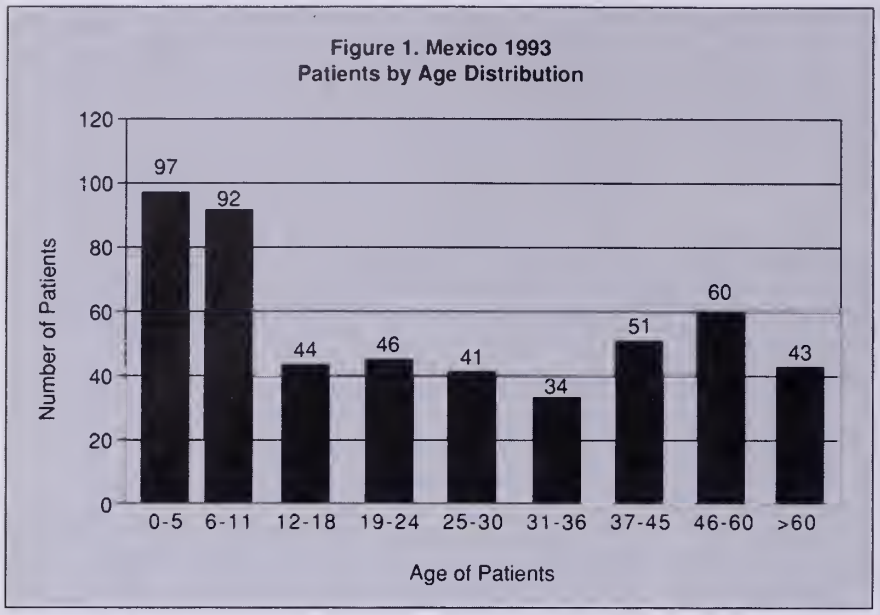
Figure 1 illustrates the age distribution of the patients seen during this trip.

The physicians, nurses, and support staff who donated time and expense to ensure this project's success have a right to feel good about our participation. We came as caring people to a neglected corner of the world and brought hope and healing. Mere words cannot do justice to the overwhelming show of gratitude

Table 2. Number of Clients Given RX per Drug**

Actifed	4
Amoxicillin	72
Ampicillin	3
Ancef	1
Ansaid	2
Antifungal cream	13
Anusal suppository	1
ASA	3
Azantac	2
Bacitracin ointment	2
Benadryl cream	1
Dimetapp	6
Doxycycline	13
Dristan	15
Dyazide	1
Ear drops	10
Ethinyl Estradial	1
Expectorant	2
Fergon	14
Flagyl	10
Floxin	1
Hismanal	1
Hydrocortisone cream	21
Ibuprofin	48
Indomethacin	14
Kaopectate	8
Keflex	12
Kwell	2
Macrodantin	1
Maalox	24
Prednisone	1
Premarin	2
Pen VK	12
Reserpine	1
Rocephin	1
Tagamet	4
Tylenol	57
Ultracef	11
Valium	1
Vermox	150
Vitamins	222
Xylocaine gel	1

**Not the total number of drugs dispensed.



and affection by our new Mayan friends. Their smiles and hugs were enough to soften the hearts of those disenchanted by present-day American medicine. Most of our team plans to return to Mexico this month, when we renew friendships in Kantunilkin.

Anyone interested in learning more about this project can contact Dr. Edde at 4400 Will Rogers Parkway, Suite 105, Oklahoma City, OK 73108, (405) 943-7162.

The Authors

R. Richard Edde, MD, is an anesthesiologist in private practice in Oklahoma City. Sandi White, RN, MS, is a nurse at Baptist Medical Center, Oklahoma City, and clinical professor at the University of Oklahoma College of Nursing.

The Oklahoma medical team prepares to leave Kantunilkin.

(From left) Team members Sandi White, RN; Joy Quinn, RNC; and Roger D. Quinn, MD, take a brief break.

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State's durable medical equipment claims now going to regional carrier

Adding to the almost continually changing health care environment, the Health Care Financing Administration (HCFA) has consolidated durable medical equipment (DME) processing into four regional DME carriers for Medicare. HCFA has named four regional carriers, with Palmetto Government Benefits Administrators being responsible for Oklahoma.

The fundamental responsibility of these four durable medical equipment regional carriers (DMERCs) is to provide the Medicare DME claims processing previously provided by 33 "local" Medicare carriers. William Toby, Jr., former acting director of HCFA, said: "With fewer carriers, experienced personnel will process claims more accurately, and reduce variations in coverage decisions. Furthermore, four carriers can more easily cross-check claims for possible fraud."

Carrier consolidation promises to (1) assure consistency of medical policy for DME throughout the nation, an especially important benefit for national suppliers; (2) assure that medical policies for DME reflect current medical science; (3) create systems to target fraud and abuse through focussed medical review; and (4) attempt to "de-hassle" the DME acquisition process for both physician and patient.

Specifically, HCFA has charged these DMERCs with developing medical policy regarding the top 100 DME items by 1991 Medicare expenditure. These DME items constituted approximately 1.3 billion dollars of Medicare expenditures in the 1991 reference year, compared to a projected 2.3 billion dollars of Medicare expenditures for DME, prosthetics, and orthotics for 1993. In addition, the DMERCs consolidated carrier "local codes," developed plans to investigate cases of fraud and abuse, and examined techniques to improve efficiency of the DME claims processing.

Transfer of the local carriers' workload to the DMERCs began October 1, 1993. Operationally, present-state Medicare carriers have been transferring their DME workload, on a monthly state-by-state basis, to the DMERCs since then, and the work should be completed by the end of January.

Significantly, with the transition, Medicare no longer bases DME payment on the state where the DME order is taken, but rather on the location of Medicare beneficiary residence.

The four DMERC medical directors completed the task of developing regional DME policy for the top 100 DME items last July. Physicians, suppliers, and manufacturers regularly met with the DMERC medical directors. The DMERC medical directors and advisors "extracted" the essence of local carrier policies (when they existed), refined them to reflect consensus medical opinion for use, and responded to input on their draft medical policies from representatives of medical, supplier, Medicare and Medicaid carrier, and manufacturer constituencies. During the comment period, Palmetto Government Benefits Administrators alone received over 2600 responses. What emerged from this process were 46 medical policies, consisting of approximately 785 separate codes for durable medical equipment and supplies.

The DMERC medical directors then proposed modifications to medical policies affecting ostomy supplies, parenteral and enteral nutrition, and wheelchairs. In creating these regionally consistent polices, they relied on patient groups' suggestions of the usual monthly requirements of quantity of ostomy supplies; outpatient nutritional policy based on expected ability to maintain or improve functional capacity; a prosthetic policy predicated on predicted achievement of a joint patient/physician/prosthetist functional state;

(continued)

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Durable medical equipment *(continued)*

and an expansion of home infusion therapy coverage to recognize the improved quality of life experienced by end-stage heart failure patients who await cardiac transplant.

These medical policies affect not only pharmacy, home health, and hospital-based DME suppliers, but also physicians, in two important ways. First, physician knowledge of the new DMERC guidelines is essential to assure that ordered medical equipment is covered as a Medicare program benefit. Second, it is estimated that eighty to one hundred thousand of this country's six hundred thousand physicians bill for DME claims and are therefore categorized as DME suppliers. The latter is especially important since all physicians who choose to file DME claims on behalf of their patients must obtain a new DME supplier number from the recently created National Supplier Clearinghouse.

Essential to the success of the DMERC's challenge is the proper utilization of 11 certificates of medical necessity (CMN) by the health care delivery team. Departing from earlier narrative based CMNs, the new standard-appearing CMNs are objective in nature, using yes/no and graded-scale responses. Using their discretion as carriers, the DMERC medical directors propose allowing the CMNs for prosthetics and orthotics, parenteral and enteral nutrition, and ostomy supplies to be completed by a member of the patient's health delivery team and *reviewed* and approved by the attending physician. Employing this new CMN structure and implementation scheme, the DMERCs balanced the requirement of accurate patient medical information to process the claim with a potential "hassle-factor" for physicians and suppliers. For electronic claims billing, claims entry screens allow electronic entry of CMN information. Paper CMNs, physician prescriptions, and supporting documentation for DME items must infrequently be submitted to the DMERCs. Documentation, as specified in the particular medical policy, must, however, be available in the physician's, physical therapist's, hospital's, and/or supplier's files for possible post-payment audit.

Palmetto Government Benefits Administrators has been designated as the Region C (which includes Oklahoma) durable medical equipment regional carrier. Claims should be directed to Palmetto GBA-N, PO Box 100234, Columbia, South Carolina 29202-3234. The toll-free telephone number is 1-800-289-1417. JJ

CAPSULES

Clinton plan doesn't address medical liability

President Clinton's health care reform package fails to effectively address the medical liability issue, according to AMA President Joseph T. Painter, MD, as he addressed the House Committee on Energy and Commerce's subcommittee on health and the environment and subcommittee on commerce, consumer protection, and competitiveness. In those November presentations, Dr. Painter said the Clinton plan fails to impose "monetary limits on awards for pain and suffering," which, he added, "is the single most important factor in containing medical liability costs." The AMA supports a $250,000 cap on noneconomic damages. Dr. Painter also noted the bill fails to adequately address the issue of restricting attorney contingency fees; it merely caps them at one-third of the award—the amount now taken in most liability cases. "This is virtually no limit at all," he said, suggesting that a sliding scale be set instead. Currently, lawyers, expert witnesses, and administrative staff get 60% of awards, leaving patients with only 40%. For effective reform, the AMA proposed the panel consider using the liability model adopted in California and Indiana, where premium costs have been successfully lowered. This approach, said Dr. Painter, ensures patients adequate compensation and discourages "frivolous litigation" as well as reduces the time required for claims resolution. He noted that increasing premiums and the threat of malpractice suits are causing physicians "to abandon practices and certain services" throughout the U.S.

AMA membership climbs after Clinton speech

By November 15, approximately 300 physicians had joined the AMA after receiving its all-physician mailing that followed President Clinton's September 22 address to the nation on his health care reform proposal; membership applications were continuing to come in. The mailing went to more than 700,000 physicians and medical students across the nation. The AMA also reported that as of November 11, almost 28,000 physicians had contacted the association, wanting to become directly involved in its reform activities through monetary contributions and grass roots lobbying efforts.

(continued)

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Call for Resolutions

All resolutions to be presented to the Oklahoma State Medical Association House of Delegates Annual Meeting must be received in the OSMA executive offices no later than thirty (30) days prior to the meeting. This year's meeting will be April 15-17, 1994, at Shangri-La Resort.

County medical societies or individuals wishing to submit resolutions should mail them to OSMA, 601 Northwest Expressway, Oklahoma City, OK 73118. Should you need assistance in drafting such resolutions, please contact the executive offices.

Resolutions must be submitted on or before March 15, 1994

Capsules *(continued)*

Volunteers needed for Doctor of the Day

Doctor of the Day volunteers are still needed to work at the Oklahoma State Capitol in February, April, and May. Physicians work approximately 9 AM to 4 PM and are assisted by a Nurse of the Day. All Doctors of the Day are introduced to the Legislature by their respective representatives in the House and Senate. To volunteer, call Bobbie Brown, OSMA staff, 405-843-9571 or 1-800-522-9452.

Dr. Keith Clark wins otolaryngology award

Oklahoma City otolaryngologist-head and neck surgeon Keith Clark, MD, is among 37 such specialists nationwide to receive the American Academy of Otolaryngology-Head and Neck Surgery Honor Award. The award was presented during the academy's 97th annual meeting in October. Established in 1934, the award recognizes those who have contributed service to the academy without remuneration, such as presenting a course or scientific paper or

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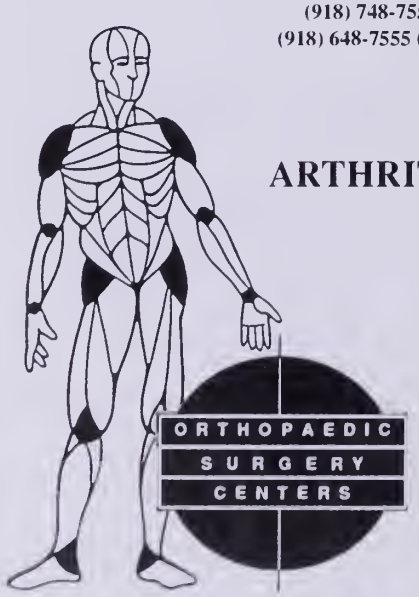

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Sen. Nickles, we count on you

Dear Senator Nickles: I had the opportunity to watch your participation in the Health Care Forum in Kansas City, Missouri, on C-SPAN on October 29th.

I applaud your support of the following:

1. Multiplicity of available health plans, each matching individual and personal needs (400 different types of plans in the room you spoke to seemed about right considering the variety and needs of the audience and speakers).

2. Discouraging oversight and/or supervision by Washington of anything resembling a global health care budget.

It is abundantly clear that Washington has bungled the following health care plans:

1. Medicare
2. Veterans Administration
3. Military Health Care
4. Indian Health Service

This is not to say that *states* do not bungle as well. Consider Oklahoma's Medicaid program which is essentially bankrupt, which does not pay for my Medicaid patient's visits to my office, which does not pay for their outpatient endoscopies, and which does not pay for their laboratory services.

This is neither an opportunity to point fingers nor to throw stones.

A balance of power is appropriate with ample room for a successful, vibrant *private* health care system which has existed up until now and which shows every sign of responding to the challenges of the moment.

President Clinton's plan as presented would establish a vast bureaucracy which would not replace, but would add to the regulatory bodies already in existence. I agree with opponents and with pundits who assert that more layers for decisions and approval will increase overall administrative costs.

Senator, millions of U.S. jobs and the American competitive position are at stake in this debate. Kindly do not ignore the votes, contributions, and people of Oklahoma and America in your deliberations. Please remember that we count on you to reject bad laws in the making and to *not accept* mediocre ones. Please support the American Medical Association and Oklahoma Medical Association's programs—Health Access America/Health Access Oklahoma—and prevent the sabotage of the most successful health care system in the world.

In the debate to follow, if there is a place where Clinton or other health reformers would like to experiment with health care, I strongly recommend that the experiment be contracted in Arkansas first.

—B. Jeffrey Wallis, MD
Lawton

CAPSULES (CONTINUED)

participating on a committee or faculty. To date, more than 100 academy members have received the award. Dr. Clark is an associate professor of otorhinolaryngology at the University of Oklahoma Health Sciences Center. He is also an adjunct professor in the Department of Speech and Communication Disorders.

Concerned Doctors of America run TV spots

In late November, the Oklahoma City-based Concerned Doctors of America sponsored several thirty-second spots on television. The announcements reminded the public that physicians are caring, dedicated professionals. Persons wishing to comment on those announcements are asked to write or call OSMA Associate Director Mike Sulzycki at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571 or 1-800-522-9453.

Dr. K.A. Miller to fill higher education post

Dr. Kimball Austin Miller, associate professor of medicine at the University of Oklahoma College of Medicine-Tulsa, has been selected to serve as a consultant-evaluator for the Commission on Institutions of Higher Education of the North Central Association of Colleges and Schools. He was one of 94 persons added to the commission's corps of 626 evaluators.

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Urinary incontinence is common

Urinary incontinence is emerging as a significant problem limiting the quality of life for many people. It appears to be more prevalent in older adults, but it is not a normal, natural aspect of aging. The problem is seen in 15 to 20% of the total population. In the literature, 50% of all nursing home placements are caused by urinary incontinence.

The Oklahoma State Department of Health's Chronic Disease Service has received a Centers for Disease Control and Prevention demonstration grant to address this condition. The project has three phases: Phase One is assessment of the target populations; Phase Two will be implementation of intervention strategies; and Phase Three will be evaluation. To effectively address the goals of this grant, the Chronic Disease staff has two partners: the University of Oklahoma College of Public Health, to assist with assessment and evaluation phases, and the University of Oklahoma College of Medicine's Geriatric Education Center, which will help with the intervention phase. The partners have developed a Coalition to Combat Urinary Incontinence, which includes specialty groups of physicians, organizations who work with older adults, and the general public.

The grant targets the general public, people with urinary incontinence, and health care professionals. The study will focus on women 65 years of age and older living in Canadian, Cleveland, Logan, and Oklahoma county communities. A physician's survey was recently completed using a randomized, stratified sample of 275 allopathic and osteopathic physicians in the specialty areas of family/general practice, urology, internal medicine, and obstetrics/gynecology within the four project counties. One of the questions in the survey was on continuing medical education. Of the physicians responding, 75% stated they would attend a symposium on urinary incontinence.

To address both the question and the response, a half day (morning) CME-accredited seminar will be held January 24, 1994, at the Oklahoma City Marriott. The featured expert will be Ananias Diokno, MD. He is the co-chair of the expert panel on urinary incontinence convened by the U.S. Department of Health and Human Services, which published *The Clinical Practice Guideline on Urinary Incontinence for Adults.*

A public survey assessed a random sample of community-dwelling women 65 years of age and older who

(continued)

IN MEMORIAM

1992

Harlan Thomas, MD	June 30
Mark Donald Vanderslice, MD	July 13
William Branch Renfrow, MD	July 21
Jared Leigh Bryngelson, MD	August 14
Jo Ann Haynes, MD	August 14
Jack Allen Harder, MD	August 19
Kenneth Lee Peacher, MD	September 20
Antone Cosmo Fina, MD	September 25
Joseph Reid Henke, MD	September 28
Charles Morris Gunn, MD	October 10
George Caarreiro dos Santos, MD	October 12
John Graham, MD	November 10
John Frederick Kuhn, Jr., MD	November 11
Roy Willie Anderson, MD	December 8
Eugene Arthur Durso, MD	December 28

1993

Howard Smotherman, MD	January 11
Robert Charles Mayfield, MD	January 9
Maud Staveley-O'Carroll, MD	January 16
David Lloyd Bell, MD	January 23
William Ray Turnbow, MD	February 28
Waldo B. Newell, Jr., MD	February 16
Homer Clark Wheeler, MD	February 18
William Alfred Waters, MD	March 19
William Niebuhr Weaver, MD	March 20
Willie George Long, MD	March 22
William Finis Ewing, Jr., MD	March 31
John Robert McInnis, MD	March 31
Chester Randall Seba, MD	April 4
Edward Cotter Murray, MD	April 9
Eric McLain White, MD	April 23
John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10

HEALTH DEPARTMENT (CONTINUED)

resided within the four counties and who had no communicative or cognitive impairment. A randomized, stratified sample of older women, whose names were purchased from Metromail Corporation, was assessed. Of the 42% responding to the survey, 17% had urinary incontinence. The age range of women surveyed ranged from 65 to 91 years, with a mean age of 73. One of the questions assessed was the need for general information on urinary incontinence. The response to this was overwhelming, regardless of whether or not the woman had a problem.

To answer this response, a multiple-channel public awareness campaign will be conducted from January through March 1994. Women with urinary incontinence who live in the target area will be invited to participate in a behavioral and educational intervention. Any interested physician wanting to participate in this project should contact the OSDH Chronic Disease Service at (405) 271-4072.

It is anticipated that valuable insights into the emerging problem of urinary incontinence will be achieved through this planned study and future research projects.

DEATHS

Kevin James Cammack, MD
1958 - 1993

Midwest City radiologist Kevin J. Cammack, MD, died December 4, 1993, in a Logan County plane crash. Dr. Cammack was born in Iowa Falls, Iowa, and graduated from the University of Iowa College of Medicine in 1985. He completed his internship and residency training at the University of Oklahoma Health Sciences Center in 1991. Dr. Cammack was chief of staff-elect at Midwest City Regional Hospital.

John Daniel Capehart, MD
1921 - 1993

OSMA Life Member John D. Capehart, MD, died in his home in Jenks on November 20, 1993. Dr. Capehart earned his medical degree from the University of Oklahoma School of Medicine in 1946. During World War II he served on active duty with the U.S. Navy. Dr. Capehart was a former chief of staff at Hillcrest Medical Center in Tulsa and also had held elective positions at Doctors and St. John hospitals. A general practitioner, Dr. Capehart practiced in the Tulsa area throughout his career. He retired from private practice in 1973.

William Best Thompson, MD
1919 - 1993

William Best Thompson, MD, a 1943 graduate of the University of Oklahoma School of Medicine, died in Oklahoma City December 10, 1993. An Oklahoma City native, Dr. Thompson completed his internship in New Rochelle, NY, and his internal medicine residency at Scott and White Clinic in Temple, Texas. From 1944 to 1946 he served on active duty with the U.S. Army Medical Corps. Upon completion of a fellowship in Boston in 1951, Dr. Thompson returned to Oklahoma City to establish a private practice in cardiology.

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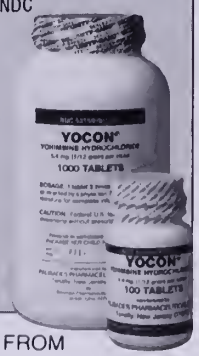

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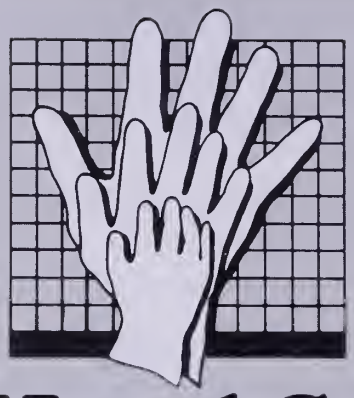

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Reprints

Authors will receive reprint order forms from the Transcript Press, PO Box 6440, Norman, OK 73070-6440, with their galleys. Other requests for reprints must be made to the Transcript Press within 30 days after publication.

Campaign Against Family Violence

Domestic violence is an ugly reality for many women, children, and the elderly in this country, and one that has long been treated as a private matter. Now, through the care, concern, and work of many individuals and groups, including the AMA Alliance, there are many programs offered which facilitate positive solutions for these victims of abuse.

This year the AMA Alliance has again made the Campaign Against Family Violence its primary goal. The Alliance has been active in encouraging both state and county auxiliaries or alliances to educate the public about domestic violence, to sponsor projects to support victims, and to develop a list of community resources for physicians.

In following the national goal, the Oklahoma State Medical Association Alliance has made the Campaign Against Family Violence their Health Promotions Project for this year. One of the first projects initiated to promote this campaign and focus on youth involved the purchase of the book *Little People's Guide to the Big World* by Trevor Romain. This book was commissioned by the Texas Medical Association Alliance to commemorate their 75th Anniversary and is a warm and entertaining book that allows children the opportunity to understand and discuss issues which might become problems. The book is designed to educate children in an entertaining way while offering adults a vehicle to discuss many topics which trouble young children, such as divorce, moving, AIDS, and others. These books were distributed to several county groups and drew such an enthusiastic response that the Oklahoma County Medical Association Alliance has adopted the sale and distribution of this book as a part of its Health Promotions Projects.

At the September meeting, the Oklahoma State Medical Association Alliance had as their speaker a member of the Child Abuse Response Center in an effort to educate their members concerning child abuse and neglect. Oklahoma County is busy preparing for the Teenage Health Symposium which will be held on January 27 from 9:30 AM to 1:30 PM for Oklahoma City and Edmond school children in grades 6, 7, and 8. Other counties in Oklahoma have had programs dealing with domestic violence or are busy implementing projects that deal with this problem as it affects their own communities.

These are but a few of the efforts that show how our state and county organizations are working to address the complex problem of domestic violence. But much more needs to be done. Every time we turn on our televisions or read the newspapers, we are confronted with examples of domestic violence.

We can prevent these situations from becoming patterns in the lives of the people in our community. We can make a difference. The resources are available. Get involved. For further information contract Cheryl Baker, State Health Promotions Chairman, at 405-341-0338.

THE LAST WORD

■ **Notable quotes from the AMA's health care** reform update meeting December 5 in Dallas:

"Let the President do a trial run of his plan in Arkansas and let it run for five years. Then after five years, if anybody still lives in Arkansas, we might have a look at his plan."—Sen. Phil Gramm (R-TX)

"It is not illegal or immoral to tell your patients what you think."—Rep. John Edward Porter (R-IL).

OSMA President G. Lance Miller, MD, reporting to the OSMA Board of Trustees after the Dallas meeting, urged Oklahoma physicians to remain "calm, cool, collected, and courteous in what will be a long, agonizing debate over health system reform.... This is a marathon, not a sprint. If we argue the case for our patients with honor, grace, and dignity, who knows, maybe even Mike Synar and those like him who honestly disagree with us on health care reform now will be won over to our side."

■ **If you have a special interest in a particular** piece of legislation, or become aware of potential legislation that could affect Oklahoma physicians, contact one of the co-chairs of the OSMA Council on State Legislation and Regulation: Larry L. Long, MD, Oklahoma City, 405-239-7134, or C. Wallace Hooser, MD, Tulsa, 918-494-1600. Correspondence to either may be sent c/o OSMA.

■ **The AMA's National Conference on Family** Violence: Health and Justice is set for March 11-13 in Washington, D.C. The invitational symposium will focus on how the health and justice systems can effectively work together to address family violence. Invited speakers are U.S. Attorney General Janet Reno, HHS Secretary Donna Shalala, and U.S. Surgeon General Joycelyn Elders, MD. Some 100 national health, justice, social service, and advocacy organizations will cosponsor the conference. Three hundred health care and justice system professionals are expected to participate. Work groups will consider gaps and barriers in the health care and justice systems and make recommendations that can be implemented at the local level.

■ **"Leadership for Medicine in Transition" is** the theme of the AMA's 1994 National Leadership Conference set for February 11-13 in San Francisco. Participants will be joined by 900 leaders from all areas of organized medicine and a variety of health care organizations to discuss critical political, economic, and social issues. U.S. Surgeon General Joycelyn Elders, MD, has accepted an invitation to speak at the conference. Five general sessions will offer information on the health cost problem, the role of public health departments in reforming the health system, the details of the White House reform legislation, and the extent of its impact.

Breakout sessions will focus on prevalent public health problems as well as specifics of state health reform and ethical issues confronting physicians. The sessions are designed to provide practical information for community use. Optional seminars will be offered to improve communications skills and learn ways of managing finances after retirement. Seminar participants also will receive up-to-date information on the latest legislative issues and learn about changes in continuing medical education.

For more information or to register, call 1-800-262-3211.

■ **The Fall 1993 issue of the state health** department's *Epidemiology Bulletin* is devoted to rabies. Detailed statistics about rabies in Oklahoma are included, as well as helpful, basic information. In 1992, March, April, and May were the peak months for both specimen submission and bite exposure incidents, with skunks being the primary carrier and dog and cat bites the most common potential exposure. Rabies is on the increase in Oklahoma, with the number of positive specimens in 1992 up 26.6% over 1991 and 65.9% over 1990. For additional information, or a copy of the bulletin (Vol. 93, No. 3), contact the OSDH. Subscription to the quarterly publication is available by request at no charge from the Epidemiology Service, OSDH, 1000 NE 10th Street, Oklahoma City, OK 73117-1299.

■ **The *1994 Oklahoma State Medical Association Directory of Physicians*** has been published and OSMA members received their complimentary copies in December. Additional copies are available to members at $25 per copy and non-members at $50 per copy. Bulk orders of ten or more are $25 per copy.

Any errors in physician listings should be reported to the association via the correction form on page 187 of the directory. A list of corrections will be compiled and published in the JOURNAL at a later date.

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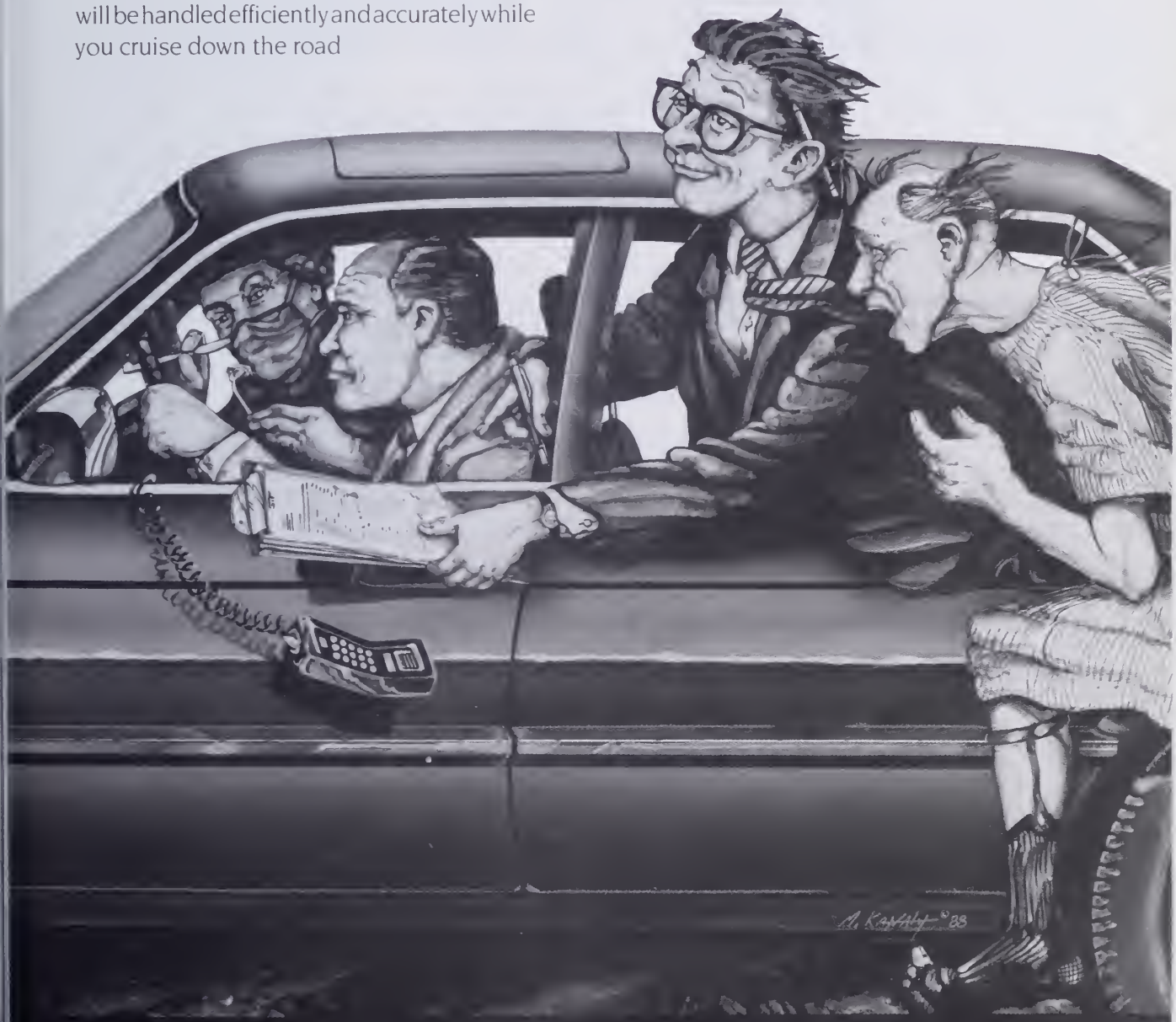

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

National advertising representative: State Medical Journal Advertising Bureau, Inc., 11437 West 106th Street, Overland Park, KS 66214, (913) 888-8781, 1-800-788-2360,

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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ABOUT THE COVER

Sunset on a farm near Okarche. Photograph by James S. Gerber, MD, Okarche. Art direction by Graphic Art Center, Oklahoma City.

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EDITORIAL

Ray V. McIntyre, MD

Back to the Marketplace!

Health care in the United States is generally agreed to be the best in the world, but it must be revised; the political time has come to renovate our health care system. Clearly, rededication and change are overdue, but what principles shall we follow to erect a new edifice amid the shambles?

In 1787, the United States needed a Constitutional Convention to bring political order out of the chaos of the Revolutionary War. In 1994 our society needs a similar convocation of mental and spiritual strength to guide Congress in health care reform.

Scientific medicine and technical progress are generally satisfactory, and the crisis arises from government regulations, an aging population, and medical insurance mechanisms.

A vital principle to establish is the need for free market medical economics. The patient must reclaim a decisive role in determining the type and amount of medical services to be provided. It is too often said: "The Agency doesn't authorize that treatment," or "my insurance won't cover that procedure." The government agencies and the insurance industry have usurped too many payment decisions. The patient's medical needs have been abrogated to government agency goals and to insurance company contracts. We must return to a free market medical economy so the patient can decide who gets paid how much.

As patients return to power, the role of medical insurance in our society will be redefined. As it is, many citizens are defrauded on insurance against ordinary daily medical expenses, as administrative costs negate the insurance principle. We should change the income tax laws to remove the corporate business tax deduction for medical insurance premiums, and then allow all taxpayers a real income tax deduction for medical expenses. A tax-sheltered medical savings account could be used effectively for rare and catastrophic losses. The concept that health care expenses are different and off budget and thus should be paid by some third party is a socialist residual that requiring new insight. The consumer pays eventually for what is consumed. In a biological system, every individual except the babies must justify their own existence.

In constructing a health care system, "cost containment" is an antithetical concept that cannot become a principle. All health care costs *something;* "there ain't no free lunch," and the true goal is efficient money use. Health care costs will naturally increase so long as technical improvements, population increases, and inflation are present—probably forever.

When we think about the efficient use of public and private patient funds, the question of medical necessity arises. The definition and determination of medical necessity is no problem when a patient spends pocket money on treatment recommended by a physician freely chosen by that patient. Difficulties arise then medical necessity is defined by an accountant bureaucrat or insurance clerk who has never attended medical school.

Patient biological variability and the context of disease profiles can markedly affect medical necessity decisions. Society needs to think through how physicians provide satisfactory treatment to diverse patients, and then construct a system that promotes that. A second principle must be established whereby medical appropriateness decisions will not be in the hands of the government, but will remain the choice of the patient and his physician.

One of the difficulties of the present system is widespread and erratic cost shifting, where one group of citizens is taxed or overcharged to pay for the medical costs of another group—the most notorious being large hospital charges on insured patients to pay for indigents in the emergency clinic. Another flagrant example are the higher taxes on the wage earner to pay for Medicare, although many patients are more prosperous than the young wage earners forced to pay the medical expenses.

Proposals that force small businesses to pay premiums for employees' medical insurance are a ploy to shift costs from the consumer to the business customer. If enacted into law, this will increase the cost, but not the value, of services and goods sold by those small businesses, and will trick yet another consumer group into thinking a third party will pay for their health care.

Reforming health care is a complex task, and every physician should give thought to the principles necessary to produce a workable system. These thoughts should then be communicated cogently to the Representatives, Senators, State Legislators, and government. Our government will not do it right without physician help.

Ray V. McIntyre, M.D.

President's Page

G. Lance Miller, MD

The Write Thing

It's not burning up I-44 between Tulsa and Oklahoma City. It's not the extra pounds gained at county medical society dinner meetings or legislative receptions. It's not the time away from practice and family visiting with physician groups across the state, attending Board and Council meetings, or representing the Oklahoma State Medical Association at national forums.

The toughest thing about being president of the OSMA is writing these darn President's Pages. There are so many topics and so little time. And there really is such a thing as writer's block.

So this month I'll take a little vacation. Rather than harangue you on the trials and tribulations facing physicians and what to do about them, I'll use this space to offer some random thoughts on what's right about medicine and physicians in Oklahoma.

Oklahoma is a great place to practice medicine. Let's never lose sight of that. We are able to practice state of the art medicine in modern, well run hospitals. We are supported by and many of us received our training from the University of Oklahoma College of Medicine, an outstanding institution that deserves more national recognition than it receives.

The unity of Oklahoma physicians provided the strength for the OSMA to produce the Physicians Liability Insurance Company (PLICO) which offers us not only reasonable professional liability premiums but even more importantly the security of knowing that our physician-run company will always defend an innocent physician rather than settle a case because it the easiest thing to do. And PLICO Health provides many of us first-rate health insurance. It is comforting to know that this physician-run health insurance company operates solidly in the black. This experience and success in the health insurance industry is particularly important now as the OSMA continues forward in the effort to create a Managed Care Organization.

The dedication of Oklahoma physicians is another good thing about medicine in Oklahoma. While there always could be more, literally hundreds of physicians volunteered thousands of hours to work on OSMA committees. The OSMA Board of Trustees, the Ad Hoc Committee on Managed Care, and the Health Access Oklahoma Task Force worked nights and weekends to study the formation of a Managed Care Organization and develop the health care reform plan Health Access Oklahoma to make sure that the OSMA will be positioned properly for any new medical environment.

If we keep the unity and dedication that have always been the hallmark of medicine in Oklahoma during these tense times of foreboding change, then our state will remain a great place to practice medicine. We can do it. We must do it.

G Lance Miller, MD

Liver Transplantation Following Preoperative Closure of Intrapulmonary Shunts

Osama Salem, MD; Vincents J. Dindzans, MD; Judith Freeman, MD; Thomas O'Dorisio, MD; Frederick Ruthardt, MD; David H. Van Thiel, MD

Intrapulmonary shunts producing basal resting hypoxemia and necessitating the continual use of supplemental oxygen by two cirrhotic men were closed prior to liver transplantation with octreotide acetate, a somatostatin analogue. The closure of these shunts was monitored by serial blood gas determinations and shunt estimations using two different techniques. Partial closure of the shunts with preoperative octreotide acetate administration allowed liver transplantation to proceed with successful engraftment and eventual permanent closure of the shunts. Currently, both patients are alive and well with normal liver function and blood gases and, most important, have no requirement for supplemental oxygen.

OCCASIONALLY PROFOUND HYPOXEMIA IS PRESENT in cirrhotic patients, who have large intra-pulmonary shunts.[1-4] Such shunts have been considered to be a contraindication for liver transplantation (OLTx) because post-operatively inadequate oxygenation in such cases has contributed to an unacceptably high rate of post-operative mortality.[5,6] Two cases are reported in which octreotide acetate, a long acting synthetic analogue of somatostatin, was used to close such shunts prior to successful liver transplantation.

From the Departments of Surgery, Medicine and Anesthesia, University of Pittsburgh School of Medicine, the Endocrine Division Ohio State University and the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma. This work was supported in part by grants from the NIDDK 32556 and NIAAA 06601.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

Case 1

Mr. P.E. is a 42-year-old male with a 10-year history of chronic liver disease occurring as a result of alpha-1-antitrypsin deficiency having an SZ phenotype. Physical examination revealed numerous spider nevi, a small liver, splenomegaly, encephalopathy, and asterixis.

Case 2

Mr. G.P. is a 46-year-old male with a history of a recently recognized liver disease diagnosed as cryptogenic cirrhosis. Physical examination revealed numerous spider nevi, an enlarged liver, splenomegaly, encephalopathy, ascites, and asterixis.

Special Studies and Methods

Because of severe resting hypoxemia, pulmonary function studies were done, including a carbon monoxide diffusion capacity and an assessment of the right-to-left shunting estimated by measuring the arterial blood gases after inhaling 100% O_2 for 20 minutes.[1,7-9] An intravenous radionuclide estimation of the degree of intrapulmonary shunting was obtained also.[10,11] Additionally, blood was obtained under basal fasting conditions and following octreotide acetate administration for measurement of plasma glucagon, a well recognized vasodilator. Following documentation of shunt closure, both individuals were transplanted and octreotide acetate therapy was discontinued.

Twenty-four hours after liver transplantation, additional fasting blood samples were obtained for

measurement of plasma glucagon. A week after liver transplantation, the radionuclide shunt study, measurement of plasma glucagon levels, and standard pulmonary function testing procedures were repeated. Two and five weeks (and also three weeks for Case 2 and twelve weeks for Case 1) after successful liver transplantation, the degree of intrapulmonary shunting was determined by measurements of arterial pO_2 before and after breathing 100% O_2.

Results

The results of pulmonary function testing procedures obtained before and after octreotide acetate administration and one, two, and five weeks after successful liver transplantation showed no significant change in the measured pulmonary volumes and diffusion capacity.

The fasting plasma levels of the glucagon before and 30 and 180 minutes after the administration of octreotide acetate, and one day, one week, and two weeks (three weeks [Case 2] and five weeks [Case 1]) after liver transplantation are shown in Figure 1.

The degree of intrapulmonary shunting assessed utilizing the nuclear perfusion technique showed that the shunt fraction was 17% (Case 1) and 14% (Case 2) of the cardiac output initially and dropped to 3% (Case 1) and 2.6% (Case 2) following a week of octreotide acetate administration (Fig. 2). One week after liver transplantation and discontinuing the octreotide acetate, the shunt was 12% (Case 1) and 14.6% (Case 2), but fell to 6% (Case 1) and 3% (Case 2) at two weeks, and to 0.8% (Case 1) and 2.9% (Case 2) by five weeks, and to 1.6% by twelve weeks (Case 1). The degree of shunting using the inhalation of 100% of O_2 for 20 minutes showed an initial shunting of 24% (Case 1) and 14% (Case 2) which decreased after liver transplantation to 9% (Case 1 & 2) after two weeks and 4.4% (Case 2) after three weeks, and 8.6% (Case 1) and 6.1% (Case 2) after five weeks, and to 8% (Case 1) after twelve weeks.

Discussion

Many pulmonary vascular abnormalities have been described in liver cirrhosis. These include intrapulmonary shunts,[1,12] portopulmonary shunts,[2,3,13,14] pleural shunts,[15] pulmonary hypertension,[10,17] impaired pulmonary vasoconstriction in response to hypoxia,[18-20] and ventilation perfusion mismatching secondary to either or both impaired hypoxic pulmonary vasoconstriction or an increase in the airway closure pressure.[21] Shunting between the portal venous system and the superior vena cava system

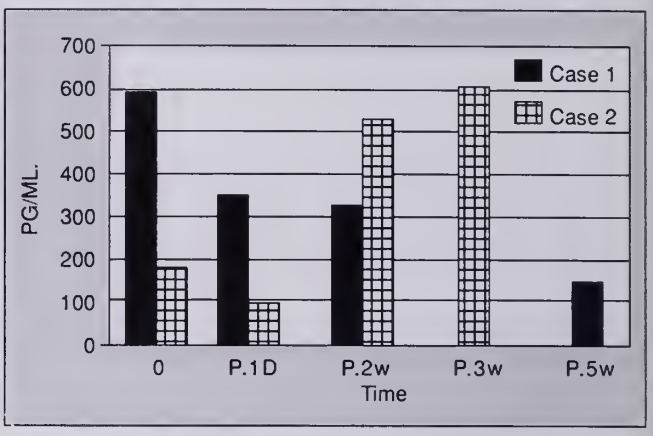

Figure 1. Plasma glucagon levels before and after octreotide therapy. O—level before octreotide; P.1D—after 1 day of octreotide; P.2W, P.3W, and P.5W equal 2, 3, and 5 weeks after initiating therapy and transplantation.

and the pulmonary venous system has been demonstrated by postmortem vascular injection methods.[2]

Until this report, severe resting hypoxemia (PaO_2 less than 50 mmHg on room air) was considered to be an absolute contraindication for liver transplantation, at least at some centers.[5,6] Two such patients known to the authors had liver transplants because of hepatic disease occurring as a result of alpha 1 antitrypsin deficiency and have died in the intensive care unit after months on a respirator not being able to be extubated postoperatively because of continued hypoxemia despite an otherwise successful liver transplant.

Plasma levels of many gastrointestinal hormones such as glucagon and insulin are elevated in the plasma of cirrhotic individuals.[22-29] The increased plasma levels of these substances in mixed venous blood has been attributed to either portal venous blood bypassing the liver through portosystemic shunts, diminished inactivation of the material because of the severity of the liver disease, or hypersecretion of the hormones and polypeptides.[30-32] Elevated glucagon levels have been shown to occur as a consequence of portosystemic shunting and to contribute to the hyperdynamic cardiovascular status of cirrhotic individuals as a consequence of its vasodilating activity.[33] This effect over years in some cases may lead to the creation of clinical manifestations of the pulmonary shunts that lead to hypoxia in a minority of chronic cirrhotics.[33,34]

It has been shown previously in patients with cirrhosis that octreotide acetate administration causes a diminution of hepatic blood flow, mesenteric blood flow,[35] and esophageal variceal pressure.[36] The suggested mechanism for these effects has been an in-

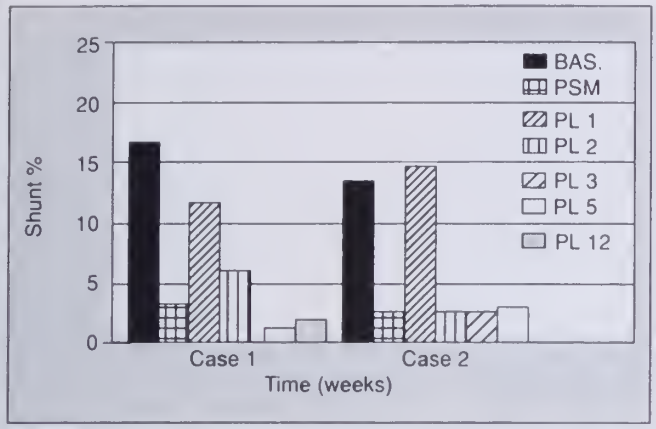

Figure 2. Degree of right-to-left pulmonary shunting determined in the two cases studied under basal (bas) circumstances and after level of octreotide (psm) and after 1, 2, 3, 5, and 12 weeks following transplantation (PL-1 through PL-12).

hibitory effect on the secretion of vasodilatory gastrointestinal polypeptides such as glucagon.

No significant change in pulmonary volume measures or diffusion capacity was evident following OLTx in the two cases reported. Nonetheless, the intrapulmonary shunt fraction declined.

The nuclear perfusion scan technique used to estimate the degree of shunting in this study is a sensitive and specific method.[37] It documented closure of the shunts one week following octreotide acetate administration with a reduction in the shunt fraction. It should be noted that during the 1 to 2 weeks post-OLT period without octreotide acetate, shunting was evident after OLT and without octreotide acetate but resolved with continued graft function. It is possible that the hemodynamic effects of octreotide acetate may have systematically effected our measures of shunting using the nuclear perfusion technique. If such was the case, one would have been expected to have lead to an overestimate rather than an underestimate of the degree of shunting determined using the radionuclide method utilized. J

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The Authors

Osama Salem, MD, is an associate professor at the University of Alexandria, Egypt.

Vincents J. Dindzans, MD, is a clinical associate professor at the Medical College of Wisconsin, Milwaukee.

Judith Freeman, MD, is an associate professor at the University of Pittsburgh.

Thomas O'Dorisio, MD, is a professor of medicine at Ohio State University

Frederick Ruthardt, MD, is in private practice in Unionrown, Penn.

David H. Van Thiel, MD, is medical director of the Oklahoma Transplantation Institution and director of the Hepatology and Digestive Disorder Center at Baptist Medical Center of Oklahoma, Oklahoma City.

Tuberculosis in a Tulsa High School

Glyn G. Caldwell, MD; Jarqulian Lee, LPN; James E. Hammarsten, MD; Janice Cox, RN; Phyllis Hicks, RN;

Following the discovery of a high school student with communicable tuberculosis, the Tulsa City-County Health Department skin tested 1,005 persons in the family, community, and school. Of the 931 persons tested at the school, 10 were reactors in March and 33 more were reactors in May. All 5 family members and 4 community contacts were tuberculin reactive. In all, 52 new tuberculin reactors were identified, evaluated, and considered for treatment or prophylaxis.

This situation illustrates the conundrum that tuberculosis remains an important public health issue because of the possiblility of transmission from small pockets of diseased persons to the larger community, especially in institutions of all kinds. Physicians and public health agencies must be vigilant and identify new cases for treatment and begin contact tracing to minimize the spread of tuberculosis.

IN EARLY FEBRUARY, 1992, A LOCAL HIGH SCHOOL STUdent was referred to the Morton Health Center for a persistent, productive cough. At Morton, the student was found to have an abnormal chest radiograph and referred to the Tulsa City-County Health Department (TCCHD). Diagnostic evaluation at the TCCHD revealed a Mantoux skin test of 17 mm, the chest radiograph showed a cavity in the left upper lung field, the acid-fast stained sputum smear demonstrated bacilli compatible with *Mycobacterium tuberculosis* and subsequent sputum cultures grew *M. tuberculosis,* which was susceptible to all first-line drugs. The patient was treated with rifampin, isoniazid, pyrazinamide, and pyridoxine, and excluded from school until repeat sputum cultures no longer contained viable *M. tuberculosis.*

An epidemiologic study of the family, school, and community contacts was begun in early March and completed in early summer.

Materials and Methods

Because *M. tuberculosis* can be transmitted through droplets generated by speaking, singing, sneezing, and coughing, especially in close quarters found among families, schools, community institutions, and occupational settings, skin test surveys were done to ascertain whether or not transmission had occurred. The TCCHD tuberculosis unit contacted all family members for Mantoux testing. In collaboration with the Tulsa Department of Education School Nurses, the TCCHD skin tested all students and school staff for tuberculosis. TCCHD staff also sought community contacts for Mantoux testing.

Mantoux testing was done by injecting 0.1 cc of purified protein derivative intradermally into the volar surface of the forearm. The tests were read between 48 and 72 hours. Any induration of 5 mm or larger was considered positive and further diagnostic evaluation performed.

Diagnostic evaluation consisted of a medical history for pertinent symptoms, a chest radiograph and, when appropriate, collection of sputum for bacteriologic culture and determination of drug susceptibilities.

From the Tulsa City-County Health Department.
Direct correspondence to Glyn G. Caldwell, MD, Director, Tulsa City-County Health Department, 4616 East 15th, Tulsa, OK 74112.

All persons found to have evidence of infection only were given isoniazid for preventive therapy. Persons with evidence of tuberculosis disease, either by chest radiograph or positive culture, were treated with isoniazid, rifampin, pyrazinamide, and pyridoxine.

Table 1. Results of Tuberculosis Skin Testing in the Family — February 1992

Person	Age	Sex	Skin Test	Chest X-Ray	Sputum Smear	Sputum Culture
Father	47	M	+	Negative		
Mother	42	F	+	Negative		
Index Case	18	F	+	+	+	+
Sibling	20	F	+	+	Negative	+
Sibling	14	F	+	Negative		
Sibling	11	M	+	Negative		

+ = Either a positive Mantoux Test, abnormal chest radiograph (x-ray), acid-fast bacteria in sputum smear, or *Mycobarterium tuberculosis* isloated in culture.

Table 2. Results of Mantoux Testing in the High School March and May 1992

Type	Persons Number March	Persons Number May	Positive March	Positive May	Negative March	Negative May
Students	793	688	10	27	783	661
Teachers	67	60	0	3	67	57
Other Staff*	71	50	0	3	71	47

*Includes bus drivers, teachers' aides, volunteers, maintenance and office personnel.

Results

The family of the patient was seen at the TCCHD and all five family members were skin test positive. A sibling of the index case was also found to have *M. tuberculosis* in sputum culture (Table 1).

The students and staff of the high school were skin tested twice, first in March and again in May, before summer break (Table 2). Of the 43 Mantoux test reactors who were not previously known to be tuberculin positive, 37 were students, three were teachers, and three were other staff members. Thirty-three of these individuals were definitely infected recently and had converted to positive between March and May. The likely sites of exposure and infection are shown in Table 3. In addition, 10 persons with reactive Mantoux tests were identified prior to this outbreak and had received preventive therapy.

Community contacts also were sought for screening. Table 4 shows these results.

Of the 52 persons who completed the diagnostic evaluation, 51 were treated. Only one adult refused isoniazid preventive therapy.

Table 3. Probable Location of Exposure and Infection for Persons with Positive Mantoux Tests at the High School March and May 1992

Location	Number of Persons (N=43) March	May	Total
Same Class	4	8	12
Following Class*	3	5	8
Bus	2	0	2
Unknown	1	20	21

*In a classroom the hour after the case was in that room.

Table 4. Results of Index Case Community Contacts Mantoux Testing 1992

Location	Persons Tested	Number Positive New	Number Positive Old	Number Negative
Church	40	4	2+	34
Other	29	0	0	29

+ Includes one person with history of Bacillus-Calmette-Guerin immunization.

Discussion

The epidemiologic follow-up of this case illustrates a number of factors about *M. tuberculosis* infection, disease and transmission. First, the index case was foreign-born, and although tuberculosis has been increasing in all populations, the majority of cases have occurred among racial/ethnic minorities.[1] Second, 86% of cases in children less than 15 years of age have occurred among racial/ethnic minorities[1] and the disease is increasing in all age groups except the over-65.[2] Third, frequency of occurrence of tuberculosis has increased throughout the United States since 1985, and this trend is continuing.[2,3] Similarly, while most countries worldwide are experiencing an increased occurrence of tuberculosis,[4] Oklahoma has experienced a slight decrease: 277 reported in 1988 and 206 in 1991, but in 1992 there was an increase, with 216 reported cases (unpublished data). Tulsa

County data is somewhat more erratic because of small numbers, but there have been 24 more cases in 1992, compared to 1991 (49 vs. 25 cases) (unpublished data).

Although tuberculosis was clearly on the wane from 1955 through 1984, the amount of effort necessary to continue this decline was eroding and had been diverted into other programs.[4] Because of this erosion, increased homelessness and crowding in shelters, more refugees and immigrants from high-prevalence countries, and increasing numbers of persons affected by drug abuse and the human immunodeficiency virus, a distinct upturn in tuberculosis incidence has occurred.[2,3]

It is increasingly important that physicians and other health practitioners, especially those caring for persons from other countries or persons affected by poverty or other diseases, especially HIV infection, be particularly cognizant of the potential for *M. tuberculosis* infection in their patients. The groups of persons shown to be at increased risk for tuberculosis are shown in Table 5. These groups frequently become pockets of disease and infection, and sources of transmission in the larger community. The situation requires on-going communication, timely reporting, and consistent collaboration to prevent further spread of the disease and development of drug resistance.

In summary, the contact investigation of a single case of communicable tuberculosis in a high school student resulted in the identification of 43 individuals at the school, 4 in the community, and 5 in the family, including one case of secondary disease. Whether the index case was responsible for all the infections cannot be determined because the entire family may have been infected in their country of origin, and the degree of contact with the source case among the school and community people tested was variable.

Table 5. Persons with Increased Risk for Tuberculosis (TB)

- Contacts of infectious tuberculosis cases
- Human immunodeficiency virus infected persons
- Persons born in high TB-prevalent countries
- Low-income populations, especially the homeless and high-risk minorities
- Intravenous drug users and alcoholics
- Residents of long-term care facilities, jail and prison inmates
- Individuals with medical conditions which increase the risk of TB

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The Authors

Glyn G. Caldwell, MD, is the director of the Tulsa City-County Health Department (TCCHD).

Jarqulian Lee, LPN, is a public health nurse with the TCCHD. She has worked with the Tuberculosis Control Program for 22 years.

James E. Hammarsten, MD, is an internist in Tulsa and is the tuberculosis consultant for the TCCHD.

Janice L. Cox, RN, is clinic manager for Communicable Disease Control at TCCHD and has 12 years' experience with tuberculosis control.

Phyllis Hicks, RN, is a public health nurse with TCCHD. She has 3 years' experience in tuberculosis control and 18 years in mental health.

SPECIAL

Surgery for Angina Pectoris: A Historical Perspective

Deborah McCollum, MD

Coronary artery disease continues to be the major cause of death in the United States each year with close to 1 million people dying of this disease annually, one third of those being under the age of 65.[1] An estimated 5 million people in this country suffer some degree of disability from angina pectoris.

ANGINA PECTORIS WAS FIRST DESCRIBED BY WILLiam Heberdon before the Royal College of Physicians in July of 1768, later being published under the title "Some Account of a Disorder of the Breast." This is a portion of his original description:

> But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris.
>
> It consists of a paroxysmal sensation of oppression in the thorax, usually under or middle sternum brought on by exertion, persisting for a few minutes and frequently radiating to the arm.[2]

The first surgical attack on angina was interruption of the nervous pathways of the heart. This was suggested in 1899 by Francois Frank, a professor of physiology in Paris, who proposed a cervical sympathetic denervation.[3] On April 2, 1916, Dr. M. Thomas Jonnesco performed the first operation undertaken for the relief of angina, denervating the last two cervical ganglia and the first thoracic ganglion on the left. The patient was a 38-year-old who is reported to have had relief from his angina.[3]

Walter Coffey in San Francisco became a proponent of this operation and in 1932 reported results of sympathectomies performed for angina in 5 patients. He cited 1 death for a 20% mortality rate, and the other 4 patients had subjective improvement in their symptoms.[4]

By the mid 1930s, three methods had evolved to denervate the heart:

1. Cervicothoracic ganglionectomy
2. Posterior rhizotomy
3. Perivertebral alcohol block of the upper sympathetic ganglia.

Operative mortality was 8 to 10% with 75% of patients reporting good results in terms of pain relief, increase in work capacity, and improvement of general condition. However, it was recognized that denervation did not improve survival from events relating to coronary artery disease, and many clinicians believed that denervation might be detrimental by destroying the protective alarm mechanisms of cardiosensory nerves.[5]

In 1939, Mercier Fauteux devised an operation on which he reported in 1946, in which all nerve fibers to and from the coronary arteries were resected at the origin of the coronary vessels, including sympathetic, sensory, and vagal fibers.[6] It became apparent that the afferent pathways from the heart arose from the thoracic as well as cervical ganglion and to completely denervate the heart meant removing the entire stellate ganglion down to the fourth thoracic

Direct correspondence to Deborah McCollum, MD, P.O. Box 26901, Oklahoma City, OK 73190.

ganglion bilaterally. With the magnitude of these procedures and the development of other techniques, denervation fell out of favor.[4]

The second operation advocated for the treatment of angina was total thyroidectomy. Hermann Blumgart did studies using radionucleotides to measure the velocity of blood flow in normal subjects and patients with altered thyroid function and, based on this, proposed surgical induction of hypothyroidism to decrease cardiac workload and subsequently decrease angina. Dr. David Berlin was the surgeon who worked with Blumgart.[5]

In the *Annals of Surgery* in 1937, Parsons and Purks tabulated a series of 133 total thyroidectomies done for the relief of angina with an operative mortality of 4%. They reported excellent results in 55% of patients and moderate improvement in 28%.[7]

Blumgart and others later used radioactive iodine to induce hypothyroidism. This method of treatment of myocardial ischemia gradually fell into disuse because of the disabling consequences.[5]

The surgeries for both cardiac denervation and thyroid ablation were done without prior testing in experimental animals, as both were undertaken with the sole objective of relieving pain.

The third operation used to treat coronary artery disease was designed to increase coronary blood flow. It was performed by Claude Beck at Western Reserve University on February 13, 1935, and reported the same year. The patient was a 48-year-old man who had previously been a farmer, but had to retire because of his severe angina. Beck made an incision to the left of the sternum with removal of the third, fourth, and fifth cartilages. After the pericardium had been opened, the epicardium was roughened with a burr. The pectoralis major was then mobilized by freeing its insertion and cut in an S shape, producing two pedicle flaps which were sutured to the parietal pericardium. The medial parts of the pectoralis and the intercostal bundles were then sutured to the parietal pericardium.[8]

This operation, or variations of it, was performed on 30 patients at Western Reserve University between February 1933 and April 1938. By June of 1940 there was a 33% mortality rate with a 65% improvement in the survivors. An autopsy done in 1 of the patients did show direct communications between the graft and the coronary circulation.[6]

At the same time that Beck was performing pectoral muscle grafts, Laurence O'Shaughnessy at the Lambeth Cardiovascular Clinic in London was studying omental flaps. In January 1936 he performed this operation on a 64-year-old, going through the fifth intercostal space and dividing the fifth and sixth costal cartilages near the sternum. He then opened up the diaphragm and explored the abdomen. Omentum was pulled up and sutured to the surface of the heart. This patient bled to death on the eighth post operative day from a large chronic duodenal ulcer.[9] By 1938, O'Shaughnessy, along with Davies and Mansell, had performed 11 cardio-omentopexies with 3 deaths and the 8 survivors showing some improvement in their symptoms. Other grafts tried at this time included pedicle grafts of skin and subcutaneous tissue, lung, and small intestine.[6]

The direct attempts to improve the blood supply to the myocardium were continued by Beck in the 1930s by the creation of pericardial adhesions in the expectation that such adhesions would carry blood vessels from the pericardium to the myocardium. A variety of chemical and physical irritants was tried, including talcum powder, asbestos, and phenol. Because poudrage was a simple operation and carried a low mortality, this procedure continued to have some proponents as late as 1960.[4]

DURING THE SAME TIME period Fauteux showed that if the coronary vein was ligated, dogs could survive ligation of the descending branch of the left coronary artery. In April 1939 he ligated the cardiac vein of a 54-year-old man with a 5-year history of angina. The patient remained asymptomatic for the next two years of follow-up.[10]

Along similar lines, Wearn in the mid 1930s showed in experiments with dogs that inducing a stenosis of the coronary sinus conferred some protection from the effects of acute coronary artery occlusion.[3]

In a combination of procedures, the Beck 1 operation began, which combined abrasion of the pericardia and epicardia, asbestos poudrage, and partial ligation of the coronary sinus. This was thought to increase coronary perfusion by elevation of the coronary venous pressure.[3] This was later modified into a two-stage procedure known as a Beck 2, in which partial coronary sinus occlusion was followed a few weeks later by creation of an arteriovenous fistula between the aorta and coronary sinus by use of a free brachial artery graft.[11] This operation enjoyed some popularity for a short time, but was never widely employed in the surgical treatment of coronary artery disease.

In 1939 at the suggestion of Freschi in Italy, Drs. Zoja and Cesa-Bianchi ligated the internal mammary arteries of a patient. This was performed under a

local anesthetic with the objective of enhancing the collaterals between the internal mammary artery and the coronary arteries. The patient, who had had multiple previous myocardial infarctions, did well post operatively and after two years was alive without evidence of additional infarctions. Fieschi had previously shown a pathway of collaterals existing from the pericardial-phrenic branch of the internal mammary artery to the coronary circulation, and hypothesized that if the mammaries were occluded at the second intercostal space, there would be an increase in blood flow through this pathway.[5]

In 1957 Glover introduced bilateral internal mammary artery ligation to the U.S. He and his associates performed this operation on 92 patients with symptomatic improvement in 68% of the first 50 patients with early evaluation. Only 42% had documented objective improvement. This operation lost popularity after double blind investigations showed similar results comparing this procedure to sham operations.[5]

In 1946, Arthur Vineberg at McGill University implanted the left internal mammary artery into the left ventricle of a dog in a tunnel, and showed that this would eventually form small anastomotic channels with the coronary circulation. Vineberg placed a traction suture of #10 cotton at the most distal point of the projected tunnel, parallel to the coronary vessels. Proximal to this, the epicardium was incised with a scalpel. A mosquito clamp was then passed through the epicardial opening through the myocardial wall for a length of one inch. The tunnel was widened by spreading the clamp.[12] In 1955 Vineberg and Buller reported on a series of 45 patients who had received their tunneled implants up to four and a half years earlier. Many had subjective improvement in their symptoms, but there was a 20% mortality rate.[13]

William Sewell at the VA hospital in Oteen, North Carolina, modified this technique by using the entire pedicle of vein, artery, and muscle for implantation. He constructed the myocardial tunnel with a knife and made 4 to 8 openings in the distal embedded artery as potential sites for collateral development. On his first reported 134 patients, he noted a 7% operative mortality rate with an additional 9% mortality in the first six months.[5]

The other center that used the Vineberg procedure in a large number of patients was the Cleveland Clinic. There Effer and Favaloro introduced the use of a median sternotomy incision for bilateral internal mammary artery pedicle implants and developed a self-retaining chest wall retractor for dissection. In 1967 the clinic reported its experience with more than 1,100 internal mammary artery (IMA) implant procedures. They reported a good to excellent symptomatic result in 53% of patients. Of those, 127 had postop arteriograms showing a 93% patency rate. Communications from the coronary artery to the implanted pedicle were shown in 54%.[14] There were also descriptions by different investigators of use of splenic artery and right gastroepiploic artery, and Sabistion and Blalock in 1959 reported using the carotid artery as a modification.

A MAJOR TECHNICAL PROBLEM in the evolution of surgery was blood vessel anastomosis. In 1902 Alexis Carrel described the trifurcation technique of vessel anastomosis, allowing direct joining of small blood vessels. Carrel, who had come to the University of Chicago in 1905, presented a paper before the American Surgical Association on May 5, 1910, in which he described the first recorded attempt at coronary artery surgery, performed on a dog. His operation consisted of construction of a graft between the descending thoracic aorta and the peripheral end of the left main coronary artery using a section of carotid artery that had been preserved in cold storage. He stated: "Unfortunately the operation was too slow. Three minutes after the interruption of the circulation, fibrillary contraction appeared, but the anastomosis took five minutes. By massage of the heart the dog was kept alive, but it died two hours later."[15] It was 42 years before Carrel's experiments were repeated in animals and 52 years before similar operations were performed on people.

Additional twentieth century nonsurgical advances that aided the development of cardiac surgery included the description by Einthoven in 1912 of EKG patterns in heart disease and first Forssmann's cardiac catheterization in 1929.

In 1953 and 1954, Murray and associates reported a series of dog experiments in which the first successful direct bypass to the coronary circulation was completed. Heparin, which was used in these experiments, helped maintain flow, and there was an intra-arterial stent used during completion of the anastomosis. Murray found the internal mammary artery to be too small and the subclavian too short for routine use, describing the most successes with axillary artery or carotid artery used as a free bypass graft from the aorta to the coronary artery. These authors suggested that this approach would be possible in patients, but recognized the need for coronary angi-

ography to locate the precise site of coronary stenosis and identify the state of the vessel, and to confirm patency at the end of the procedure. Because of the fear of ventricular fibrillation and the high intrinsic risk of this operation, it was not used in humans.[5]

On October 29, 1956, Bailey performed the first coronary endarterectomy, which was reported in the *Journal of the American Medical Association* in 1957. This was done at normothermia without cardiopulmonary bypass and involved opening the left anterior descending coronary artery distal to its obstruction, passing a hollow curette back up through the artery into the coronary opening in the aorta, and withdrawing a cast of the intima.[16]

Longmire, Cannon, and Katts in 1958 and 1959 reported on successful outcomes in 5 of 9 patients in whom endarterectomies were performed. Again, this was done without bypass but, differing from Baily, they opened the artery directly over the disease.[17]

The first cardiopulmonary bypass for coronary surgery was performed by Dubost and colleagues February 15, 1959, and was reported in 1960. The patient was a 39-year-old with severe angina who underwent unroofing of a syphilitic overgrowth of both coronary ostia using heart-lung bypass and profound hypothermia. The patient recovered fully and was free of angina during exercise early after operation.[18]

The first selective coronary arteriogram was performed by accident in 1958 by F. Mason Sones, Jr., who was a pediatric cardiologist at the Cleveland Clinic. He was attempting to inject a large bolus of contrast media retrograde across the aortic valve into the left ventricle. After the injection he found the dye had gone into the right coronary artery rather than the left ventricle and knew his catheter had been misplaced. Since the visualization of the right coronary was so good, and since the patient tolerated the injection well, he went on to develop the technique of selective coronary arteriography.[19]

The first direct coronary surgery guided by preop angiography was performed by Senning in 1958 and reported in 1961. Preop films showed a narrowing the proximal left circumflex and anterior descending coronary arteries. Using cardiopulmonary bypass with profound hypothermia, two saphenous vein strips were placed as on-lay grafts onto these two vessels for a length of 4 cm. Three months postop the patient was asymptomatic with heavy activity, and angiography showed that both grafts were patent.[20]

The technique of gas endarterectomy was also developed during the 1960s. With this technique, a needle was placed subadventially and CO_2 gas under pressure of 400 mmHG was used to separate the diseased intimal layers of the artery from the normal adventia.[21]

This technique was first used on coronary vessels in May 1966 by P.N. Sawyer at Kings County Hospital on a patient undergoing a mitral valve replacement. The patient had a total occlusion of a long portion of the anterior descending coronary artery and a four-and-one-half-inch plaque was removed. This patient did not survive but the technique was successful in limited subsequent cases.[6]

ANOTHER INTERESTING WAY of combating angina was by decreasing oxygen consumption of the heart by use of radiofrequency carotid sinus nerve stimulators which were surgically implanted. Electrodes were placed in contact with the carotid sinus nerves bilaterally and connected to a receiving unit buried subcutaneously. The patient could activate the stimulator with the onset of angina or prophylactically prior to beginning activity which would normally lead to angina. Stimulation would result in a decrease in blood pressure, mild decrease in heart rate, and relief of angina. This was abandoned due to a lack of efficacy.[22,23]

The March 1961 issue of the *Journal of Thoracic and Cardiovascular Surgery* reported an experimental method of internal-mammary-to-coronary-artery anastomosis using tantalum rings. This work was done on dogs at Albert Einstein College of Medicine by Robert Goetz, but was never used clinically.[24]

One of the first aortocoronary bypass grafts using saphenous vein was performed by Sabiston at Johns Hopkins, April 2, 1952, but wasn't reported until 1974. The patient was a 41-year-old man who had a proximal occlusion of his right coronary artery for which he had previously undergone an endarterectomy. A repeat operation was performed for severe angina. A saphenous vein was harvested and anastomosed to the ascending aorta using a partial occluding clamp. The distal saphenous vein was then sutured to the end of the right coronary artery, which was transected below the area of occlusion. Post operatively the patient maintained cardiac function but within a day died of a stroke which was thought to have originated as an embolus from the proximal anastomosis. This operation was performed without cardiopulmonary bypass.[5]

In 1967, Kolessov reported 5 of 6 patients who survived internal mammary artery (IMA) to coronary artery anastomosis with improvement after operation. The first of these operations had been performed

in 1964 and reported in the Russian literature in 1965. A left thoracotomy was used in all 6 patients. Preop angiography was not performed and palpation of the coronaries was needed to identify the areas of atherosclerosis and choose the site for placement of the graft. This distal part of the IMA was also tunneled through the myocardium in an effort to encourage secondary collateral revascularization as well as direct arterial inflow.[25]

The first reported use of saphenous vein for bypass of coronary artery disease in humans was by Favaloro and associates in 1969. This operation was performed May 9, 1967, on a 51-year-old woman with a totally occluded right coronary artery. On this patient, Favaloro replaced a segment of stenotic artery. Postop angiography showed the graft to be patent. This initial success stimulated the use of this operation on a large number of patients at both the Cleveland Clinic and at other institutions.[5]

Favaloro made these comments regarding left main disease prior to the development of this procedure:

> Left main trunk obstruction was certainly our worst enemy, and every time I had to face it before the development of the saphenous vein graft technique, I went to the operating room with a combination of challenge and fear in my soul, feeling certain than the chances of success were minimal.[26]

Later in 1967, Favaloro began to anastomose the saphenous vein end to end from the ascending aorta to the distal coronary artery, and in 1968 began bringing the vein into the artery obliquely, as is done today.[4]

On February 8, 1968, Bailey reported the first successful internal mammary-to-coronary-artery bypass in the United States. It consisted of a right internal mammary to distal coronary artery bypass with cardiopulmonary standby and cross clamping of the right coronary artery during construction of the anastomosis.[5]

Also in February 1968, George Green at New York University performed the first successful IMA to the left anterior descending coronary bypass. This operation was performed with bypass and with temporary occlusion of the left anterior descending at the site of the anastomosis. Three of the first 9 patients operated on died in the first month postup, and each had a myocardial infarction in areas not perfused by the single bypass. This finding led Green to combine internal mammary artery with multiple saphenous vein grafts, and subsequently the operative mortality was reduced to less than 5%.[6]

Today coronary bypass surgery remains well established as an effective treatment for patients with severe coronary atherosclerosis. This surgery is performed annually on over 300,000 patients in the U.S.[1]

Patient-related characteristics have changed with time compared to the early years, with today's patients being older, with more extensive heart disease and more comorbid conditions. They are also more likely to be women and are more likely to have had previous bypass surgery. Today's hospital mortality at major medical centers is 1 to 3%.[27]

Coronary artery bypass surgery is an anatomic treatment for the anatomic sequelae of a metabolic disease, and until we are able to intervene on a metabolic level, will remain a mainstay in the treatment of angina. ℐ

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The Author

Dr. McCollum is in her first year of cardiothoracic fellowship at the University of Oklahoma College of Medicine.

TREATMENT OF ARROW WOUNDS IN THE AMERICAN INDIAN-FIGHTING ARMY: THE TECHNIQUE OF BREVET LT. COL. JOSEPH H. BILL

Joe D. Haines, Jr., MD

Figure 1.
Col. Joseph H. Bill

The history of medicine contains many chapters with seemingly insoluble problems that were overcome by great innovators. Contributors like Pasteur, Reed, and Salk altered the practice of medicine forever. However, along with the giants of medicine, there have been legions of other physicians who have made lesser, but not unimportant contributions. Many of these discoveries involved techniques that became obsolete as technology progressed, and many others formed the basis for further medical advances. Such was the case of Dr. Joseph H. Bill's technique of treating arrow wounds in the Indian Fighting Army of 1866-1890.

THE TREATMENT of arrow wounds has become a lost art in this age of technologically sophisticated warfare. Just 100 years ago the U.S. Surgeon General's office issued lengthy circulars detailing the treatment of arrow wound victims. But with the final shot fired at Wounded Knee in 1890, the Indian Wars ended and the discussion of arrow wound treatment became an anachronism.

The resurgence of bow hunting and recreational archery increases the chance of today's practitioner encountering an arrow wound. But if nothing else, the technique of arrow removal provides a case study of how a resourceful frontier physician met a difficult challenge in the field.

Brevet Lieutenant Colonel Joseph H. Bill wrote extensively on the surgical extrication and treatment of arrow wounds in Surgeon General's reports, journal articles, and textbooks. Col. Bill was appointed Assistant Surgeon in the U.S. Army on April 3, 1860, and was assigned to the American West.

The army in which Dr. Bill served changed dramatically after the close of the Civil War. The U.S. Army began demobilization and greatly reduced the number of physicians in the medical department. By 1869, there were only 161 commissioned medical officers to staff the 239 army posts scattered throughout the southern and western plains. In order to fill the ranks, the army resorted to hiring contract surgeons, and in 1868, 282 contract surgeons were employed.[1]

To receive a commission in the medical corps of the regular army, an applicant had to pass a rigorous

Please direct correspondence to Joe D. Haines, MD, 1501 W. Rogers Boulevard, Skiatook, OK 74070.

series of written and oral examinations administered by the Army Medical Board. These exams often lasted up to six days and covered all areas of clinical and academic medicine, as well as general knowledge. Passing the Army Medical Board was considered quite a distinction since a considerable number of applicants failed.

Those who passed were eligible for a commission as Assistant Surgeon, with rank and pay equivalent to that of a first lieutenant. After five years, a man could be promoted to captain and again stand for examination to advance to Surgeon, which carried the grade of Major. In the post–Civil War period, the army was popular with many young physicians seeking adventure and fortune (usually in mining or ranching claims) on the western plains.

The work load of a frontier surgeon was anything but light, however. Besides providing medical care, the typical post surgeon served as hospital administrator, public health officer, food inspector, pharmacist, personnel director, and staff officer. The physicians faced many and varied medical problems. Most problems were a result of or related to infection, nutritional problems (including alcoholism), and trauma. The lack of fresh fruits and vegetables made scurvy common and gastrointestinal infections could immobilize entire commands. Trauma from injuries and combat required a significant portion of the surgeon's attention.

Ether and chloroform were available as anesthetics. Even though Joseph Lister had published his principles of antiseptic wound care in 1867, his techniques were not accepted in American medicine for another ten to fifteen years. Amputation was by far the most common surgical procedure, and the accepted treatment for abdominal wounds was "expectant," which consisted of "quieting the bowel" with opium or morphine.[2]

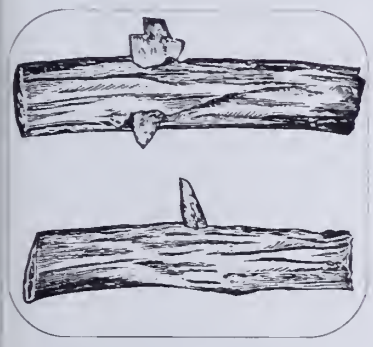

Figure 2. Ribs of buffalo transfixed by arrows

The treatment of arrow wounds presented a real dilemma for the frontier surgeon. If the arrowhead was embedded in bone, it was almost impossible to extract by the standard method, with forceps. An arrowhead in soft tissue, if manipulated the wrong way, could separate from the shaft, making its recovery nearly impossible. And since thoracotomy and abdominal exploration were discouraged at the time, wounds to the chest and abdomen were usually fatal.

Dr. Joseph H. Bill recognized the inadequacies in treating arrow wounds and set to work to resolve the dilemma. He first set out to gain a good working knowledge of the particular characteristics of each Indian tribe's arrows. He studied the physical dimensions and structure of many arrows and became proficient in judging an arrow's depth of penetration by measuring the exposed shaft and then subtracting from the known overall length. He also was able to determine the plane in which the arrowhead lay by observing the slit in the feathered end.

Figure 3. Iron arrowhead lodged in left temporal bone

In his *Notes on Arrow Wounds,*[3] Dr. Bill described the manufacture of arrows:

> The arrow is composed of two parts, a shaft and head. The shaft varies in length from two to three feet. Usually it is made from a *limb* of the dogwood tree. A limb of a quarter-inch diameter is taken, soaked in water, the bark peeled off, and then cut into suitable lengths.
>
> The straightening process is now commenced... It will often require three days to straighten a single shaft (effected by hand-twisting). A notch is now filed at one end for the bowstring, and a slit made in the other to receive the straight quadrangular stem which comes off from the base of the head. The head is made of soft hoop iron (it could also be made of stone), filed into the form of an isosceles triangle, and furnished with a stem to attach it to the shaft...[4]
>
> An arrow-head varies in length from half an inch to two inches... No two arrows are alike. The stem at the base of the arrow-head is pushed into the slit made for it in the shaft, and held there by wrapping narrow ribbons of tendon spirally around the split sides of the latter, thus clamping these together.[5]

This tendon fixation proved especially dangerous for the victim because as the tendons became wet with the fluids of the wound, they lengthened and loosened. Thus when the shaft of the arrow was pulled upon, it was very likely that the head would disengage from the shaft and be left behind. For this reason, and also due to the valuable information provided to the surgeon by the intact arrow shaft, Dr. Bill urged that the arrow remain undisturbed.

A deeply lodged arrowhead was practically impossible to extract with forceps. The procedure was also exceedingly dangerous since the operator had to blindly probe the wound for the foreign body. Great force was sometimes required to dislodge arrowheads lodged in bone. As Dr. Bill noted, "In the case of a man shot in the shaft of the humerus by an arrow, it was only after using both knees, applied to the ends of the bone as a counter-extending force, and a stout pair of tooth-forceps, that we succeeded in removing the foreign body."[6]

The case of Private Bishop vividly illustrated Dr. Bill's point:

Private Bishop was shot in the head of the humerus with an arrow, and the shaft having been plucked out, the iron head was left deeply embedded in the bone. The man was in great pain, synovia was flowing out of the wound, and all motion was lost. I enlarged the wound, introduced my finger, and so ascertained the position and depth of the arrow-head. It was very deeply implanted. I introduced forceps, seized it by its base, but could scarcely obtain any "purchase." I at last succeeded in grasping it tightly, and bracing my knees against the patient's thorax, I applied all the traction I could muster. Suddenly the arrow-head flew out of its seat, and I would have fallen on the floor, had not the steward caught me. The wound healed well. It was treated by evaporating lotions, and a rigorously antiphlogistic diet for the patient. Motion in the joint was not lost, though somewhat impaired.[7]

Since the tract of the arrow wound closed back upon itself, deep abscesses and hematomae were common complications. The upper extremity was the area of the body most often wounded, followed by the abdomen, chest, lower extremity, head, and neck. Since an expert bowman could discharge six arrows per minute, multiple wounds were the rule.[8]

In some wounds of the extremities, the point of a metal arrowhead could become bent and present a complication in removal. As the arrowhead scraped the edge of the bone, muscles of the limb could contract, bending the point of the arrowhead until it resembled a fishhook. Digital examination could easily confirm this deformity of the arrowhead. In these cases, Dr. Bill advised seizing the arrowhead with forceps, vibrating it from side to side, and pushing rather than pulling to disengage it. Rotatory movements, aided by the finger, then usually accomplished removal.

Bill strongly opposed the conservative management of arrowheads embedded in bone, since retained arrowheads so often caused infection and abscess formation. As Dr. Bill stated:

The incisions (to explore for the arrowhead) should be large and free—boldness rather than prudence governing our actions. We might as well cut the patient's limb up until we do find the arrowhead, for if it is left, amputation will be necessary, and worse than this can hardly ensue from the "cutting up" we have advised.[9]

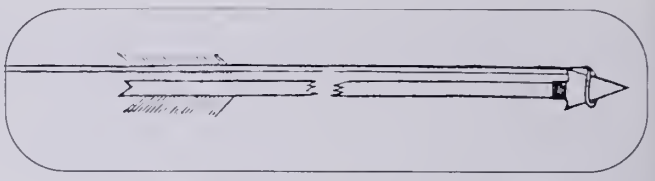

Figure 4. Wire loop for extracting arrowheads embedded in soft parts.
(After a drawing by Dr. Bill.)

In considering arrow wounds to the skull, Dr. Bill advocated gentle traction on the arrowhead; this also served to elevate an associated depressed skull fracture. Following elevation of the fragment, the wound was closed with collodion, the head shaved, and cold applied.[10] The case of Miguel illustrates these principles:

Miguel "Nigro," the post-guide at Fort Union, was shot with an arrow by a Utah Indian. I found the arrow-head sticking in the left parietal bone, the shaft having been detached. I made traction on it, and drew it out of the wound. The symptoms of compression present at once vanished, the man turned over and sneezed, and rose up on his feet. I had made arrangements to trephine the skull if necessary, but I had probably restored to its proper level that portion of the inner table which was depressed, so that measure was unnecessary. The cause of the compression was gone, and I had nothing to trephine for. The next day the man complained of headache. His face was flushed, eyes suffused, pulse hard, and irregular. I ordered croton oil, shaved his head, and applied cold. Presently, when delirium came on, I bled him until he fainted. This bleeding was repeated the night of the same day. The next day he was greatly better; the croton oil had operated well. The man was left to recover, which he did in three days.[11]

As Dr. Bill reported, arrow wounds to the chest and abdomen were usually fatal, but occasionally miraculous recoveries, like the following, were documented.

A Mexican, name unknown, whilst travelling the road near Fort Defiance, was shot by Navajoes with five arrows, all the arrows injuring the lung, and one of them passing through the upper border of the liver. I saw the man twenty minutes after the accident. The bleeding was most profuse, and the man fainted. Both lungs were wounded; I made the worst possible prognosis, but proceeded to extract the arrows, all of which I safely removed. After the hemorrhage had ceased, I applied muslin soaked in collodion to each of the wounds, eight in number, and had the patient put to bed, and given a grain of morphia. The next morning the patient was more comfortable than I had expected. He had been vomiting, and I gave him some acetum opii and acetate of lead, to quiet this and restrain secretion. In the afternoon the man complained of pain on the right side, and difficulty in breathing. I removed the dressing from the wound, involving the liver and the lung, and broke up some clots whereupon a considerable quantity of mixed fluids were discharged from the wound. These

contained bile as I discovered by Pettenkofer's test. This procedure greatly relieved him, and he expressed himself much better. I ordered the prescriptions of the morning repeated. The next day the patient vomited a considerable quantity of black decomposed blood, and complained of frequency of urination. Urine very dark coloured, sp. gr. 1036; urine contained cholic acid and sugar. All medicines ordered to be discontinued. After this the patient gradually recovered, all the wounds healing by first intention, except that implicating the liver, and that ceased discharging on the sixth day. In two weeks the man left the hospital, his urine still containing sugar, but no bile. He had passed during his illness about thirty-six ounces per diem. In a few days the last traces of sugar disappeared from his urine, and in six weeks from the day he was wounded, he returned to the post from a scout, in order to have removed a large polypus of nasal cavity, which had plagued him a great deal during his sickness.[12]

Trying to decrease the mortality rate of arrow wounds of the chest and abdomen, Dr. Bill invented two devices that were both simple and effective. One was designed for removing arrows lodged in bone and the other for removing arrows embedded in soft tissues.

The first step in removal of an arrow was to estimate the depth of penetration and to determine whether the arrow was lodged in bone. As explained earlier, the depth of penetration could be accurately estimated by measuring the length of the exposed shaft and subtracting from the arrow's known length. To determine whether the arrowhead was fixed in bone, the shaft was taken between the thumb and forefinger and gently twirled. Lack of mobility meant bony fixation.

If the point of the arrowhead was not lodged in bone, a "wire having a loop at the end, and bent at a right angle was introduced (Fig. 4), and pushed down through the wound (using the arrow shaft as a guide), until the loop was past the point of the arrowhead. By then slipping the loop over the point of this, and drawing the wire gently back, it was found from the sense of resistance that the loop had snared the treacherous body."[13]

In other words, the wire loop snared the arrowhead and gentle traction then removed the arrow without detaching the head from the shaft. If the arrowhead was lodged in bone, a different approach was required. Since the plane of the arrowhead and the notch for the bowstring were usually the same, the arrow shaft could be rocked to and fro in a direction at right angles to this plane. This approach diminished the chance of loosening the head from the shaft, and with patience, the arrowhead would sometimes dislodge.

If gentle manipulation was unsuccessful, Dr. Bill's second arrow extraction device could be employed. The device was merely a very long Coghill's suture

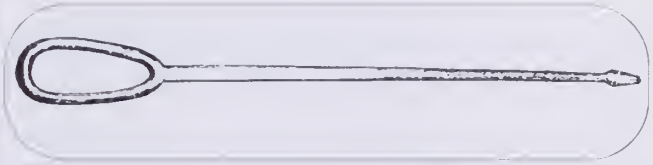

Figure 5. Wire-twister recommended by Dr. Bill. It should be twelve inches long. (From Dr. Bill's paper.)

Figure 6. Wire loop twisted once on itself for snaring impacted arrowheads. (From Dr. Bill's paper.)

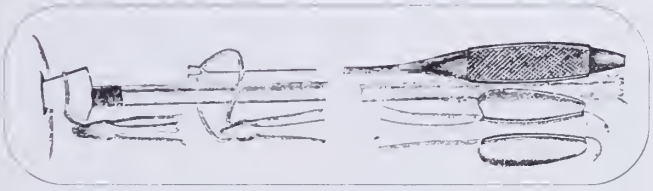

Figure 7. Application of wire loop to arrowhead embedded in bone; the loop is adjusted by aid of the wire-twister and *porte-mèche*.

wire twister (Fig. 5). By passing the ends of a two and one-half foot wire through the holes of the suture twister, carrying them up the shaft and securing the ends to the handle, a loop at the end of the instrument remained. This loop was then passed over the feathered end of the shaft and advanced until it encircled the arrowhead (Fig. 6 & 7). One end of the wire was then pulled upon to embrace the head tightly and this was followed by one or two twists of the instrument to secure good purchase on the arrowhead. Gentle traction and rocking was then applied to disengage the head.

Finally, a case submitted by Dr. C.A. Pope, professor of surgery at St. Louis Medical College, appeared in *Hamilton's Treatise on Military Surgery* that testifies to the skill, resourcefulness, tenacity, and courage of a true physician:

A lieutenant in 1860 in a skirmish with Indians in New Mexico received an arrow shot in the left superior maxilla. The spear shaped iron point, two and three quarter inches long, with a small neck for the fastening of the shaft of the arrow by means of the sinew of a deer, entered the face a little below the orbit and was completely embedded in the bone. Its direction was backwards and outwards. The surgeon of the post immediately endeavored to extract the foreign body. At first it was hoped that this might

be accomplished by forcible traction on the arrow itself. The only effect of this, however, was to detach the feathered wooden shaft from the iron headed point, which remained firmly in the bone. (You see, he violated the first principle—never make traction on the shaft.)

Various means were used in an attempt to extract the head but all failed. Slight secondary hemorrhage occurred from the nose off and on for the next few weeks. I saw him five weeks after the injury at his home in St. Louis. There was some enlargement of the left side of his face. The wound of the cheek had skinned over. A muco-purulent discharge issued from the corresponding nostril. I felt a hard point beneath the integuments of the cheek and incising the scar found the neck of the arrow head. I failed in its extraction until I used a pair of powerful forceps. A smart hemorrhage from the nostril and the wound followed. Rest, opium, cold plugging and bandage arrested the hemorrhage. A few slight hemorrhages subsequently occurred.

The case now seemed to progress favorably and he was able to go out on the streets. On a visit to my office he complained of a stiffness and inability to separate the jaws as widely as usual. I advised him to make gentle efforts to open the mouth. In less than an hour from this time his trouble commenced.

The whole cheek became hot and painful. High fever with renewed hemorrhage set in and caused me much anxiety. Extensive extravasations of blood took place, and to relieve the tension, I made free incisions both in the mouth and on the cheek and neck from which large clots were pressed out. The hemorrhage continuing at intervals, and finally on three successive nights occurring with regularity about twelve o'clock, and the patient being reduced to the lowest point of safety, I determined to tie the carotid without delay.

This was done by candlelight on the night of the 16th of December two months after the receipt of the injury. Such was the extreme condition of the patient he fainted during the operation, although he was in the recumbent position. The operation itself was a delicate and difficult one, as the parts were very much swollen and altered in their relations by infiltrations. The bleeding never recurred, and the final recovery of the patient was complete.

This lieutenant lived to fight again; years later he was killed in the Battle of Fredericksburg. He was General Bayard.[14]

At a time when most surgeons were reluctant to explore the abdomen in the case of irretrievable arrowheads, Dr. Bill was a strong advocate for abdominal exploration. As Bill stated, "Expectant treatment in such cases is really no treatment at all," and "the arrowhead must be extracted or the patient will die."[15] Bill also advocated the use of a cuirass or bullet-proof vest for protection against arrow wounds to the chest and abdomen; however, his recommendations were not employed. Col. Bill additionally developed a magnetic probe for missiles in tissue.[16]

Besides Dr. Bill's innovative techniques for arrow removal and his outspoken support for aggressive surgical management and prevention of arrow wounds, he was also responsible for educating soldiers in the field in the treatment of arrow wounds. By convincing the troops to resist the temptation of attempting to remove the arrow, the surgeon had a much greater chance of extracting an intact arrow.

Col. Bill died July 21, 1885, after suffering a massive cerebrovascular accident. But over 100 years later, Bill's advice holds true; the surgeon continues to recommend that a protruding foreign body be left undisturbed until he receives the patient.

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The Author

Joe D. Haines, Jr., MD, has a family practice in Skiatook.

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— OFFICIAL CALL—

THE HOUSE OF DELEGATES OF THE OKLAHOMA STATE MEDICAL ASSOCIATION WILL CONDUCT ITS 88TH ANNUAL MEETING AT SHANGRI-LA RESORT AFTON, OKLAHOMA

APRIL 15-17, 1994

Opening Session: 9 a.m. Friday, April 15
Closing Session: 9 a.m., Sunday, April 17

All members, delegates, alternate delegates, and county society officials are encouraged and urged to attend. Business to be brought before the House of Delegates must be submitted by March 15, 1994. All items of business will be debated in open reference committee hearings on April 15, 1993

Any member of the association may submit business for consideration by the House of Delegates. For help in preparing information for submission, please contact
OSMA headquarters
601 Northwest Expressway, Oklahoma City, OK 73118
405-843-9571 or 1-800-522-9452

MARY ANNE MCCAFFREE, MD
SPEAKER OF THE HOUSE

NEWS

OU-based *Journal of Child Neurology* earns international recognition

The *Journal of Child Neurology*, an international medical journal with editorial offices based at the University of Oklahoma College of Medicine, has reached a milestone in its nine-year history—a high ranking from the annual Institute for Scientific Information Science Citation Index, Journal Citation Reports.

The index ranks the journal as eighth of 55 international pediatrics journals, based on its recent index measurement of a 1.347 impact factor. The impact factor measures the frequency with which a journal's articles are cited by other source publications and is an indication of the high quality of articles published in that journal, says Dr. Roger Brumback, editor-in-chief of the *Journal of Child Neurology* and professor of pathology in the OU College of Medicine.

In addition, of 145 international neuroscience journals, the *Journal of Child Neurology* was rated number 81. "We are extremely pleased with these rankings," Brumback says. "They reinforce what we have known for several years—that we have one of the best neurology journals in the world headquartered at the OU College of Medicine."

Dr. Richard W. Leech, professor of pathology, serves as book review editor for the journal. Dr. Gary Bobele, assistant professor of neurology, and Dr. Betty Pfefferbaum, professor of psychiatry and behavioral sciences, serve on its editorial board.

CALL FOR RESOLUTIONS

All resolutions to be presented to the Oklahoma State Medical Association House of Delegates Annual Meeting must be received in the OSMA executive offices no later than thirty (30) days prior to the meeting. This year's meeting will be April 15-17, 1994, at Shangri-La Resort.

County medical societies or individuals wishing to submit resolutions should mail them to OSMA, 601 Northwest Expressway, Oklahoma City, OK 73118. Should you need assistance in drafting such resolutions, please contact the executive offices.

Resolutions must be submitted on or before March 15, 1994

American Medical Association issues resolutions for a healthy new year

The physicians of the American Medical Association issued their 1994 "New Year's Resolutions for a Healthier America" on December 28. The AMA provides the annual list as a public health reminder that the most effective health care is preventive care. Many of today's diseases and injuries can be controlled or avoided through changes in lifestyle or attention to hazard. The five New Year's resolutions are:

1. Quit Smoking or Assist Another to Quit

Make today the first tobacco-free day for the rest of your long life. Only ten years after quitting, a former smoker's risk of lung cancer is equal to a non-smoker. If you are a non-smoker, become an advocate

continued

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Business Manager — Cathy Devine

AMA Resolutions *(continued)*

for smoke-free work places and public areas like restaurants and shopping centers. Educate your children early on the dangers and unpleasantness of smoking before the lure of slick advertising convinces them otherwise.

2. Conduct a Home Safety Inspection

And do it with your children. Go through your home to find hazards to you and your family. Check the smoke alarms to make sure they have fresh batteries and are functioning properly; make sure all doors and windows open quickly for emergency exits. Teach your children how to use 9-1-1 and other emergency numbers.

Put safety latches on cabinets that hold medicines, cleansers, and dangerous solvents. Place safety plugs in electrical outlets; make sure electrical and curtain cords are out of the way of curious children, and be particularly careful with space heaters. Hire a heating expert to clean and inspect your furnace and water heater. To avoid accidental scalding of children, set your water heater no higher than 120°F.

3. Learn Child and Adult CPR

Cardiopulmonary resuscitation (CPR) is not just for heart attacks. CPR training includes life-saving techniques for drowning, choking, sudden loss of respiration, infant CPR, and many other situations, and can be learned in less than one day. Remember, most CPR is performed on individuals known very well by the rescuer.

Classes are available through schools, churches, hospitals, government, and park districts across the country.

4. Go Through Your Medicine Cabinet

Throw out your old medicines. Drugs lose their effectiveness and safety while sitting around the house. Never take a drug after the expiration date. When in doubt, call your physician or pharmacist for advice. *Never take a drug not prescribed for you.* Drug interactions, allergies, and the unknown spell danger for anyone taking something not intended for them. Also, inspect your cabinet for safety, making sure children are unable to reach the contents.

5. End the Cycle of Family Violence

Violence in the home has reached epidemic proportions. Despite being under-reported by women, children, and the elderly, it still kills and injures millions. Violence is a cycle that must be broken. The first step is to ask your physician for help. A violent home never improves by doing nothing; you must decide to find assistance. Physicians are trained to deal with the physical wounds of violence and will help you find professionals to resolve the causes as well. Counselors are available through local hospitals, physicians, churches, or neighborhood groups. Violent behavior is a disease, and the first step toward a cure is to seek treatment for yourself or someone you love.

In Memoriam

1993

Howard Smotherman, MD	January 11
Robert Charles Mayfield, MD	January 9
Maud Staveley-O'Carroll, MD	January 16
David Lloyd Bell, MD	January 23
William Ray Turnbow, MD	February 28
Waldo B. Newell, Jr., MD	February 16
Homer Clark Wheeler, MD	February 18
William Alfred Waters, MD	March 19
William Niebuhr Weaver, MD	March 20
Willie George Long, MD	March 22
William Finis Ewing, Jr., MD	March 31
John Robert McInnis, MD	March 31
Chester Randall Seba, MD	April 4
Edward Cotter Murray, MD	April 9
Eric McLain White, MD	April 23
John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney-Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6

Vital signs

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Health Department

Pitfalls in Metabolic Screening

The Oklahoma Public Health Laboratory tests over 50,000 newborns each year for metabolic diseases, including hypothyroidism, phenylketonuria, galactosemia, and hemoglobinopathies. Each year several thousand newborns cannot be tested with the specimens submitted due to specimen collection problems, incomplete patient information on the laboratory requisition, or improper handling and mailing. Inaccurate results or reporting create a potentially disastrous situation.

Blood collected from a heel stick is placed on an FDA-approved filter paper collection kit that contains five circles: the paper is then dried and sent to the health department for testing. To ensure that the filter paper absorbs a standard amount of blood, it must meet rigid manufacturing quality standards. All circles must be filled to provide adequate specimen for testing. Before the filter paper specimen is punched for testing with an automated punching machine, the specimens are checked for quality. Due to the risk of low results, specimens are rejected if the amount of blood placed on the filter paper does not evenly saturate or fill the circles; high results can occur if the circles are over saturated or improperly filled using a capillary tube.

Improper handling of the filter paper before specimen collection may result in contamination by disinfectants, ink, etc., and crushing or tearing of the paper can result in inadequate absorption of the blood. When the specimen is collected, the filter paper must be air dried at room temperature for three to six hours before shipping. The integrity of the specimen will be compromised if it is improperly dried, exposed to high temperatures, or held over fourteen days before shipping.

Many times specimens are collected and handled

OSMA Physician Recovery Program

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Publication of directory prompts the usual round of corrections

A small flurry of corrections inevitably hits OSMA headquarters in the weeks following the release of a new physicians directory. This year has been no exception (see sidebar).

Errors should be brought to the attention of OSMA Membership Coordinator Judy Lake so that corrections can be made in the association's computer files. They will also also published in the JOURNAL.

This year, for the first time, the OSMA directory was produced in-house on the association's desktop publishing system. Staff members Judy Lake, Kathy Musson, and Susan Records combined their skills, using output from the association's Unix mainframe computer and two personal computers to produce the directory pages. It was a crash course in database manipulation and directory production, but those involved were pleased with the results. Suggestions for improvements are being kept on file, and next year's directory should be even better (current plans call for publishing annually from now on, rather than biannually).

HEALTH DEPARTMENT (CONTINUED)

properly, but cannot be accepted because the laboratory requisition is not complete (lacks a birth date, name, date of specimen collection, or return address), does not have the correct information, or the correct test has not been selected. This information is required by the federal Clinical Laboratory Improvement Amendments 1988 and by the Public Health Laboratory for computer tracking of the data and reporting.

"Universal precautions" must be followed when collecting and preparing these specimens for shipment. The specimens must *not* be packaged in airtight, leak-proof plastic bags since that can cause incorrect test results. Ideally, the filter paper specimens should be enclosed in strong, tear-proof, air-permeable, and water-resistant envelopes. A biohazard label is not required unless there is evidence that the specimen contains an etiologic agent.

Avoiding the potential pitfalls in metabolic screening is not difficult. All it takes to assure optimal specimen integrity is attention to detail. This means making sure that all patient information is complete, and that specimen collection, handling, and mailing are properly performed.

Corrected 1994 Directory Listings

Make a note of these corrected listings in your 1994 *OSMA Directory of Physicians*, or clip and tape for future reference.

Oklahoma County Medical Society's phone number on page 5 should be 843-5619.

GILLOCK, WILLIAM ROBERT
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1979-UNIV OF OKLAHOMA COLL OF MEDICINE
7170 S BRADEN #175
TULSA, OK 74136
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JOHNSON, CRAIG S
COLON AND RECTAL SURGERY
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6565 S YALE #902
TULSA, OK 74136
PHONE 918-481-2788 FAX 918-481-2791

JOHNSON, JON MARK
INTERNAL MEDICINE
1984-UNIV OF OKLAHOMA COLL OF MEDICINE
608 S HESTER
STILLWATER, OK 74801
PHONE 405-377-8000 FAX 405-377-8040

LACKEY, JAMES D
OBSTETRICS AND GYNECOLOGY
1974-UNIV OF OKLAHOMA COLL OF MEDICINE
105 S BRYANT #401
EDMOND, OK 73034
PHONE 405-330-2229 FAX 405-340-6353

MIRANDA, AMALIA
OPHTHALMOLOGY
1978-FAC DE MED DE LA UNIV DE SEVILLA
SUITE 1010, 3435 NW 56TH
OKLAHOMA CITY, OK 73112
PHONE 405-945-4747 FAX 405-947-4748

REID, ROGER J
GENERAL PRACTICE, PSYCHIATRY
1931-MANITOBA FACULTY OF MEDICINE
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ARDMORE, OK 73401
PHONE 405-223-3219 FAX

SNIDER, GERALD A
FAMILY PRACTICE
1970-UNIV OF OKLAHOMA COLL OF MEDICINE
1500 N MAIN ST
MUSKOGEE, OK 74401
PHONE 918-683-2283 FAX 918-683-2333

TURLINGTON JAMES THOMAS
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1970-BAYLOR COLL OF MEDICINE
3314 W OKMULGEE
MUSKOGEE, OK 74401
PHONE 918-687-9700 FAX 918-687-8020

WILEY, M. DENISE
1977-GEORGE WASHINGTON UNIV SCH OF MEDICINE
3434 NW 56TH ST
OKLAHOMA CITY, OK 73112-4415
PHONE 946-5678 FAX

WILLIAMS, JOHN DAVID
INTERNAL MEDICINE
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Brutal Ironman Triathlon no match for Tulsan's determination

Hawaii's Ironman Triathlon. It's one of the most grueling events in sports, with competitors swimming 2.5 miles (in the ocean), cycling 112 miles, and running 26.2 miles... in the same day.

And Tulsa pediatrician Donna Brown, MD, has shown it's not the exclusive domain of full-time athletes. On October 30, 1993, she achieved the goal she'd set for herself some three years earlier—competing in and completing the Ironman. Her time was a respectable 14 hours, 17 minutes, and she became the first Oklahoma woman to finish the event.

Dr. Brown raises her arm in triumph (left) as she crosses the finish line 14 hours, 17 minutes after starting the long race.

Below, she manages a smile on a downhill section of the course.

For those who haven't the determination to walk around the block, nor the stamina to swim one lap in a pool, the accomplishment cannot be overstated. That it was done by a busy practicing physician and mother on her 47th birthday is truly inspiring.

Dr. Brown learned to swim just three years ago, right after a knee injury interrupted her fledgling running program. Doing laps in a swimming pool allowed her to continue her aerobic training while the knee healed. She admits it was a full month before she could complete four laps in the pool without stopping.

A new bicycle came next, and shortly thereafter, the decision to train for triathlons. When Dr. Brown completed her first swim, bike, and run competition in May 1990, the die was cast. She went on to become nationally ranked in her age group, and last May, with the help of "a good coach and lots of supportive friends," began training specifically for the Ironman.

"I love to cycle and would spend long hours on the bike to get myself ready," she says, but, "running and swimming did not come easily. It seemed every time I would build up mileage on the run, I would get injured."

However, perseverance paid off. When 1,438 of the world's best endurance athletes lined up on the Kona beach for the start of the 1993 Ironman Triathlon, Dr. Brown was among them; she was also among the 1,353 who finished.

Today Dr. Brown recalls her personal victory with relish. "The Ironman experience was such an incredible opportunity to participate in an event with the finest triathletes in the world. Everyone competing knew what it took to get to this race. We knew that each of us had sacrificed and worked hard for this opportunity.... I had accomplished a goal that few people had had the opportunity to pursue, and became included in the ranks of those who had finished.

"The sport of endurance is one that we are learning more and more about as time goes on. The body can meticulously and systematically build a base for performance beyond what the average person thinks is possible. I have learned we can *all* achieve more than the average. If we have dreams and goals and desires to be the best we can be, it can be done."

Mrs. Wilbur Lewis details husband's devastating skiing accident

Oklahoma City surgeon Wilbur C. Lewis, MD, was critically injured in a skiing accident in Colorado during the Christmas holidays. His family asked that the following letter be published in response to the flood of questions and expressions of concern that followed.

BECAUSE OF BOTH THE OUTPOURING OF INTEREST and concern and the continuing confused stories relating to the recent tragic accident of our beloved husband and father, my children and I are making public the following facts.

Account of the Accident

For many years, our family has gone to Copper Mountain, Colorado, to ski during the interim between Christmas and New Year's when the Physician's Winter Retreat, sponsored by the University of Oklahoma Health Sciences Center, features a continuing medical education forum. We arrived two days before Christmas with our children Karen, her husband, Howard; David, his wife, Sadako, and baby, Jason; Leanne, her husband, Carey; and Christen. Wilbur, an excellent skier, was coming down B slope at Copper Mountain on Monday, December 27, a bit after 12:30 PM, with Leanne, Carey, and David. The day was somewhat snowy and overcast, so there were no shadows to indicate ridges or other elevations in the terrain. Ordinarily, those places are flagged to signal to skiers.

Leanne, in front of the pack, went right and took off her skis to go in to lunch. Wilbur followed her, but turned to the left. Just a few steps from the door to the inn is a drainage ditch with a culvert into it, making a slight elevation which did not create a shadow. Because it was not flagged, Wilbur did not note its presence. He was not going very fast because he was headed toward a snow bank to remove his skis. As he skied over the area, the tips of his skis caught in the elevation and he fell full-face forward

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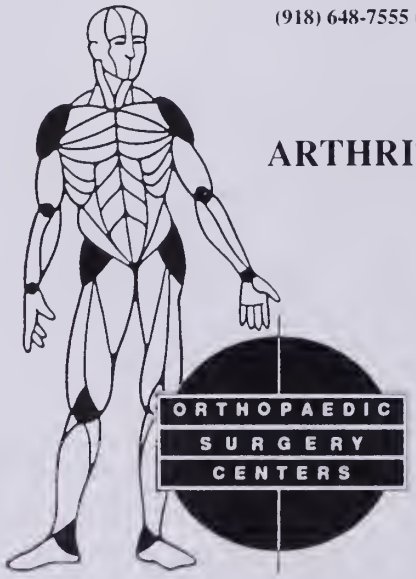

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into the ground. The impact caused a ring fracture of his first cervical vertebra and shattered the second one. His injury was the kind often associated with those which divers receive. Because that area of the spinal cord services autonomic systems of the body, such as breathing, he was immediately without the ability to breathe.

Carey saw the entire scenario, and rushed to him, calling for help. David, last in the group, came just after the fall, but watched in panic as he saw his father turning blue. Leanne ran to him and he mouthed, "Get help! Get help!" Attending our same conference were a Dallas cardiologist and his physician assistant wife who immediately began CPR and resuscitation. The ski patrol came quickly with oxygen and carried him to the nearby clinic.

"We have learned Death Valley is not in California; it is at the base of a ski slope in the Colorado Rockies."

After emergency attention, Wilbur was evacuated to Denver to St. Anthony Central, a trauma center, placed on a ventilator, and diagnosed as quadriplegic: paralyzed from the neck down. His condition was so grave that he was not expected to live through the night. However, when his vital signs and mental condition improved by Tuesday morning, his orthopedist, neurologist, and general surgeon decided to take him to surgery to do three things: fuse the damaged vertebrae to prevent further spinal cord injury; make a tracheostomy in his throat for the ventilator; and implant surgically a feeding tube in order to maintain his nutrition. After five hours on the fusion by the orthopedists, the general surgeon decided to wait until the next day to do the two additional procedures.

After his surgeries, his body continued to react to the shock he had suffered, but lights of encouragement began to appear. The most important one consists of Wilbur, himself. His mind is absolutely clear; he has no brain damage. After several interchanges between his mouthed questions and our carefully explained narratives, he knows exactly his condition and what we face. He quizzed his physicians and caregivers; I have told them he will be their greatest ally in the recovery process. We have watched him process information and assess where he is. He gave a few evidences of grief the first two days, but determination seems to have replaced any denial or anger.

His Present Condition

The reality we face is bleak, dismal, and grim. Wilbur is a ventilator-dependent quadriplegic. But we have some positive signs and reasons for hope. First, his mind is not impaired. His spirit remains gentle, good, and absolutely rock solid. We have other tiny toeholds to begin our climb back up our big mountain. From an enormous team of specialists, we have started gains. Willbur can swallow, and since four days after the accident, he eats pureed food. Because he has done well, the feeding tube is used only at night.

With increasing stabilization, some of the emergency lines and tubes have been removed. He was placed in a highly technological computerized bed which moves and turns him, preventing circulatory problems and bringing normalcy to his lower lungs which had begun to fill with fluid. His lung specialist tested his ability to move air four days after the accident; Wilbur could take in about 20% of the amount needed to inhale a breath. A changed ventilator mode works at increasing that ability, and he has been able to stay on the new pattern, although with extreme difficulty.

Wilbur experiences horrible pain. We struggle to keep his head comfortable. Because his fusion will need several weeks to mature, he must wear a rigid neck collar, one more source of distress. The effort to communicate brings unbearable frustration. The open tracheostomy means he cannot make sounds, but he can mouth his words. The day he could form the *t* and *st* sounds, he conveyed great pride. He lies with his head flat, so his visual field is very constricted. The occupational therapist brought him some prism glasses, so he could see at an angle. Leanne, Karen and I celebrated by singing and doing the motions to "Into the Air, Junior Birdmen" at the foot of the bed.

WE BASE MUCH OF OUR OPTIMISM IN TWO EVENTS: an FDA drug study, and getting into The Craig Institute for Rehabilitation. The Craig, as we hear the insiders call it, is a nationally famed rehabilitation facility in Englewood in the Denver area, less than ten miles from St. Anthony Center. Staffs from the two have a close working relationship. Two days after the accident, a doctor and two other staff members from Craig came to see me about placing Wilbur in an FDA-sponsored double blind study of Sygen, or

GM-1. He qualified because of his single injury, otherwise perfect health, and the need to begin the medication within 72 hours of his accident. As a family, we agreed to enter him in the protocol. He will have the medication 56 days and be monitored up to two years to determine the drug's efficacy before it is placed on the market. Also, The Craig has agreed to accept Wilbur as he is, on a ventilator, to begin their intensive rehabilitation program.

Our dear friend John McGraw, orthopedist from Laurel, Mississippi, came to Denver on Friday after the accident to be with me and my family. We went to The Craig to decide what we thought about it and to evaluate the near miracle stories we heard of their recovery work. We came away extremely impressed and hopeful. Wilbur will be admitted there by the time you read this information.

What We Are Doing

Wilbur will stay in Denver. We have made important contacts and connections which we believe will mean some kind of re-entry into productive life. Because we want to avoid knee-jerk emotional behavior and embrace practical decision making, we will work out a rotation scheme for our family and Wilbur's siblings to go to The Craig where small apartments for families form part of the treatment process. Wilbur is adamant about wanting normalcy to continue as much as possible, so we have agreed that we will keep our jobs, stay in school, and rotate the time spent in Denver. In addition, we plan to keep his office open.

What You Can Do

Your love, interest, and flood of concern have sustained us. They have also locked up the switchboard of St. Anthony Central. We have diligently tried to have one member of the family to speak with everyone who called. But our number present will dwindle, so I am setting up four liaison centers for four basic groups where you may call for exact, up-to-date information on Wilbur's condition.

I will contact my pastor, Dr. Gene Garrison, Dr. John Poarch, Dr. John McGraw, and Wilbur's office staff regularly to give them updates. My church, First Baptist, Oklahoma City (232-4255) will service the general public; Dr. Poarch (840-3793) has volunteered to serve as a contact with the local medical community; Dr. McGraw (601-649-5990, office) will provide information for our friends across the country; and at the office (848-8848), Betty and Annette will have information for patients. Call the office or the church any time you wish. If you do not get your information from one of these four sources, you cannot be assured that your facts are correct.

Please, do not send flowers, fruit, or other gifts. Your thoughts humble us, but small ICU rooms and hospital settings cannot accommodate them. If you so desire, you can send an equivalent amount for such expressions to one of three places where we have set up scholarships and endowments: (1) Office of Development, Oklahoma Baptist University, Shawnee, Oklahoma 74801; (2) Office of Development, Southwestern Baptist Theological Seminary, P.O. Box 22000, Fort Worth, Texas 76122; and (3) The Baptist Medical-Dental Fellowship, Brotherhood Commission, 1548 Poplar Avenue, Memphis, Tennessee 38104. Attach Wilbur's name with your gift; officials there will apply it accordingly.

The flood of prayer effort has sustained us. All kinds of spiritual energy from Jewish, Buddhist, Hindu, and Christian sources are being expended for us. With humility, we accept every bit of it as we have learned Death Valley is not in California; it is at the base of a ski slope in the Colorado Rockies.

—*Gladys S. Lewis*

Note: *Dr. Lewis is an ordained Baptist minister and 1955 graduate of the University of Oklahoma School of Medicine, He decided as a child to become a medical missionary, and he and his wife pursued that dream in Paraguay, working there as missionaries for ten years (described in Glady Lewis's book* On Earth As It Is). *When political unrest forced their return to the states in 1970, Dr. Lewis established a private practice in Oklahoma City. In addition to his practice, he continued his involvement in medical education, with a special interest in training doctors and nurses to work in South America. His numerous professional and church-related activities have earned him many honors including, last year, investature as a Knight of Malta; he was one of only ten people in the world so-honored in 1993.*

DEATHS

Fannie Lou Leney-Hayward, MD
1902 - 1994

Fannie Lou Leney-Hayward, MD, a native of Pittsburg, Tenn., died January 2, 1994, in Tulsa. She was a 1927 graduate of the University of Oklahoma

School of Medicine and began a practice in pediatric allergy in Oklahoma City in 1929. She later became an instructor in pediatrics and allergy at OU. Dr. Leney was granted Life Membership status when she retired in 1978. She moved to Tulsa that same year and married Harold Hayward. In 1983, Dr. Leney-Hayward received the Award of Merit from the American College of Allergists.

Kirk Thornton Mosley, MD
1906 - 1994

OSMA Life Member Kirk T. Mosley, MD, a native of Mayflower, Ark., died January 3, 1994. A graduate of Tulane University School of Medicine, Dr. Mosley began his medical career as a missionary to China in 1931. After training in public health, he returned to the Pacific Theater as a medical officer in the U.S. Army from 1942 to 1945. After the war Dr. Mosley taught preventive medicine and public health at the University of Oklahoma before becoming professor and chairman of the department. He held the post until 1960, when he became Oklahoma State Commissioner of Health. Dr. Mosley resigned in 1966 to work for abroad for the Ford Foundation, but returned to the state in 1975 as medical director of the Garfield County Health Department in Enid. He retired in 1991.

Henry Grady Ryan II, MD
1923 - 1993

Henry G. Ryan II, MD, died December 17, 1993, in his hometown of Norman. Dr. Ryan attended the University of Oklahoma School of Medicine, graduating in 1947. From 1948 to 1960 he maintained a family practice in Healdton, interrupted by two years' service with the U.S. Army Medical Corps, 45th Division, during the Korean conflict. In 1960 Dr. Ryan moved his practice to Lindsay, and in 1980 to OU's Goddard Health Center in Norman. There he served as OU athletic physician in 1981 and as medical director of the Goddard radiology department, director of laboratory services, and medical administrator of GHC's After Hours Clinic.

Richard Charles Wade, MD
1932 - 1964

Oklahoma City obstetrician-gynecologist Richard C. Wade, MD, died January 6, 1994. A native of Catskill, N.Y., he was graduated in 1968 from the University of Miami (Fla.) School of Medicine. He completed his ob-gyn residency at the University of Oklahoma in 1972 and established a private practice in Midwest City.

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Classified ads are 50 cents a word, with a minimum of $25 per ad. A word is one or more characters bounded by spaces. Box numbers will be assigned upon request and will add 6 words to the total. Payment must accompany all submissions. **Orders will NOT be accepted via telephone or fax.** Mail ad with payment to OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118. Deadline is the first of the month preceding the month of publication.

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"Organized medicine must serve as an advocate for patients," stressed Dr. Fullin.

The American Medical Association (AMA) couldn't agree more. We're committed to focusing physician attention on the issue of family violence.

You are invited to join Dr. Fullin and to join with him in his efforts to bring quality health care to those in need. Become a member of the American Medical Association today.

Members of the AMA are encouraged to join their state, county and specialty societies.

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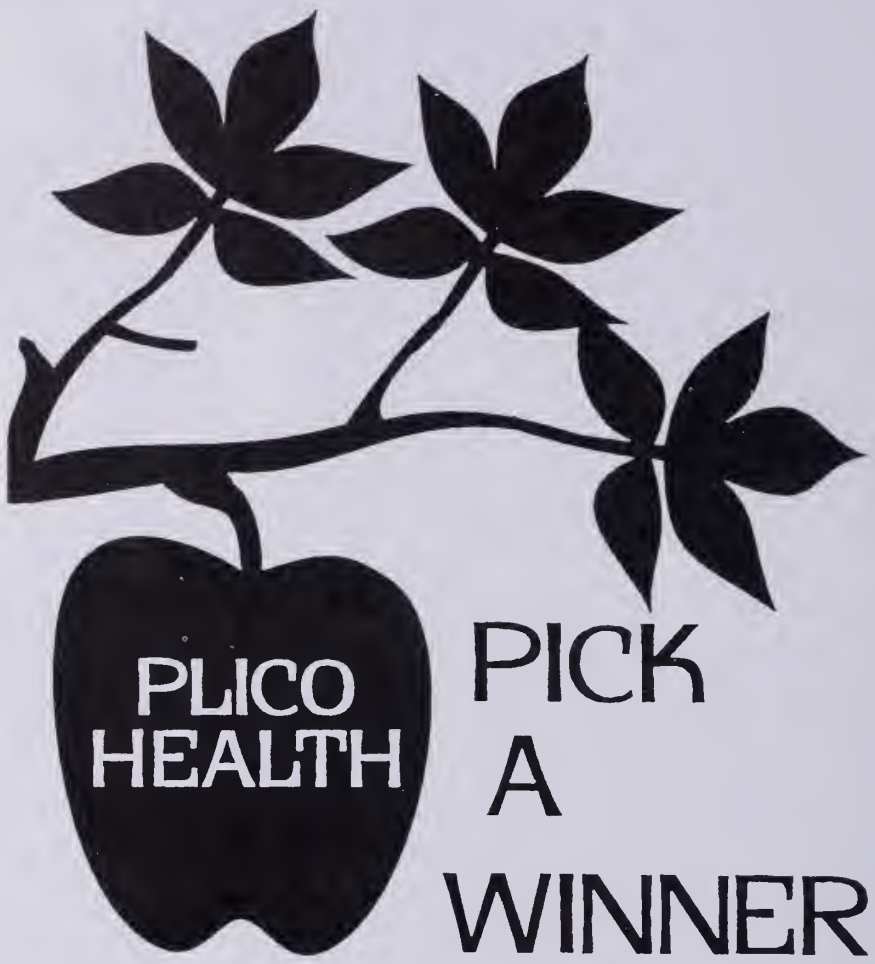

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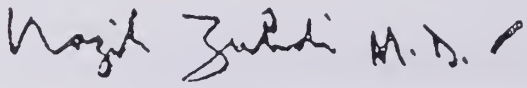

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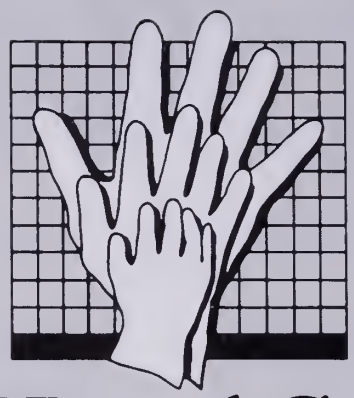

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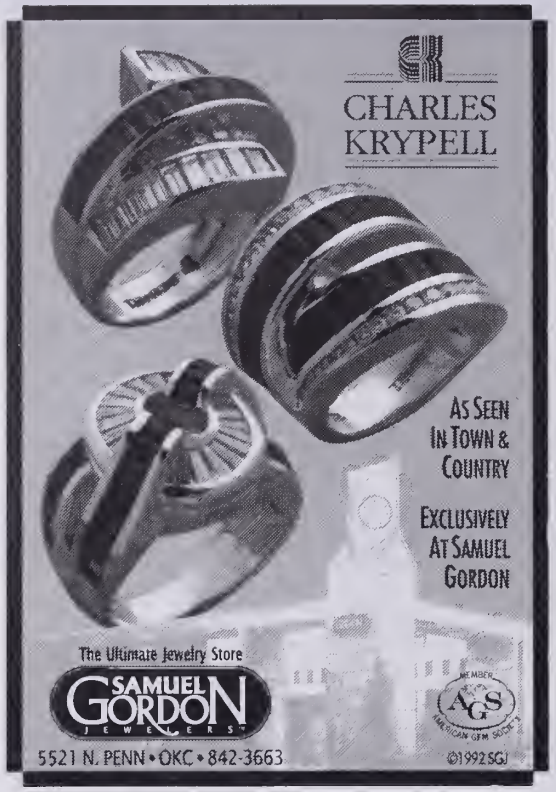

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Authors will receive reprint order forms from the Transcript Press, PO Box 6440, Norman, OK 73070-6440, with their galleys. Other requests for reprints must be made to the Transcript Press within 30 days after publication.

AUXILIARY

One Hour = $ 21.03

Since 1955, fund raising for the American Medical Association Education and Research Foundation (AMA-ERF) has been the only philanthropic effort of the AMA Alliance. For the fourth consecutive year, AMA Alliance members have raised more than $2 million for the American Medical Association Education and Research Foundation to benefit the nation's medical schools. Why is made clearest by a recipient of AMA-ERF funds, this fourth-year medical student from the University of Iowa:

"As many of you may remember, medical school is an intellectually and financially taxing period in a physician's life. I was able to prepare myself mentally for the rigors of medical school, but could not have met the financial demands without outside assistance.

"After applying for financial aid through the college of medicine, I became eligible for a loan program designed to help cover expenses with low interest loans for any financial need not met through federal programs. Without these funds, I would have been forced to apply for loans with much higher interest rates through private banks and lenders. The college of medicine has been able to provide this necessary assistance due to the generosity of organizations such as the Iowa Auxiliary. I thank you for your support and encourage you to continue your assistance in the future."

In these changing economic times, the success of fund raising efforts for the AMA-ERF are remarkable. Since its founding more than 40 years ago, AMA-ERF has distributed more than $57 million to help medical students and schools achieve excellence in education.

Funds are needed by medical schools now more than ever. Still remaining unique in that 100% of each contribution is given to the designated medical school, there are no funds deducted for administrative costs. You designate the medical school that will receive your contribution and the fund that will receive your contribution: the Medical School Excellence fund, which provides grants that are used at the school's discretion; or the Medical Student Assistance Fund, which is used by the school for direct financial aid for students.

Our fund-raising projects can make a difference, whether it be in the purchase of state-of-the-art equipment, funding student research projects and internships, or providing scholarships and loans to the students who need them.

The Alliance has chosen the "One Choice... One Hour" theme for their fund-raising efforts. For one hour of medical education in the U.S., the Foundation has calculated that the cost is $21.03. Contributing one hour, two hours, or five hours of medical education for one medical student means that the tradition of excellence demonstrated during the 40-year history of AMA-ERF will again have real importance at the most practical level. By attaching a price to a single hour of medical education for a single student, the financial difficulty of attending medical school in the 1990s becomes instantly clear.

"The availability of AMA-ERF funds has assumed greater importance in view of diminishing federal and state support for medical students' financial aid and the dramatic rise in student fees. In the upcoming 1993-94 school year, fees for first-year medical students will be more than three times the amount paid by first-year students just five years ago.... Our medical students will be informed of the tremendous support of the AMA ERF." —*Gerald S. Lazarus, University of California, Davis.*

We ask you to consider a donation of one hour, two hours, or five hours of medical education to the medical school of your choice. As one supporter put it: "Whether my child chooses to be the physician and/or inevitably becomes the patient, I will have participated in the quality of care given."

At our state convention in April we will have a Western Dance where there will be some "fun shopping" for our physicians and spouses. The proceeds from a silent auction of twenty-five or thirty baskets will go to AMA-ERF.

Your "One Choice... One Hour" donation can be sent to our state AMA-ERF Chair, Karen Ghormley, 410 BelAire, Blackwell, OK 74631.

THE LAST WORD

■ **The Physician Manpower Training Commission** moved its Oklahoma City office in January. The new address and phone number are as follows: Physician Manpower Training Commission, 1140 Northwest 63rd Street, Suite 302, Oklahoma City, OK 73116, (405) 843-5667, FAX (405) 843-5792.

■ **Oklahoma City ophthalmologist Thomas E.** Acers, MD, is recipient of the 1994 Dean's Award for Distinguished Medical Service. The award was presented by the University of Oklahoma College of Medicine at its 10th Evening of Excellence, held January 27, 1994, at the Marriott Hotel in Oklahoma City. The annual black-tie affair is sponsored jointly by the state's business and medical communities and honors a leader from each sector. Its goal is to raise research funds for the University of Oklahoma Health Sciences Center.

Dr. Acers is past director of the Dean A. McGee Eye Institute and Gaylord professor of ophthalmology at OU. Physicians previously honored are Mark A. Everett, MD, 1993; Stewart G. Wolf, Jr., MD, 1992; Robert G. Tompkins, MD, 1991; G. Rainey Williams, MD, 1990; Tullos O. Coston, MD, 1989; George S. Bozalis, MD, 1988; Gerald Rogers, MD, 1987; George Garrison, MD, 1986; and Don O'Donoghue, MD, 1985.

■ **OSMA members are reminded that March 31** is the deadline for payment of 1994 dues.

■ **All lots of Albuterol Sulfate Inhalation Solution**, 0.5%, were recalled in January by manufacturer Copley Pharmaceutical, Inc., due to microbial contamination discovered in some lots. The solution is widely as a bronchodilator by asthma patients and others with respiratory ailments. Only patients with compromised immune systems, including AIDS and cancer patients undergoing chemotherapy or radiation treatment, and persons with cystic fibrosis or chronic obstructive lung disease are at risk for potentially serious infection, said Kenneth Zack, MD, corporate medical director.

Albuterol Sulfate Inhalation Solution 0.5% distributed by the following private labels has also been recalled: Aligen, Astra, Geneva, Goldline, Major, Moore, H.L. Moore, Qualitest, Rubgy, and Schein. The recall does not affect Copley's Albuterol Sulfate Inhalation Solution 0.083%.

All pharmacists have been asked to review their dispensing records since December 1, 1991, and notify affected patients, who should then contacts their physician regarding replacement medication.

■ **The Oklahoma State Medical Association** Board of Trustees, acting as a committee of the whole, has named the Garvey Group of Merrick, NY, to serve as consultant on the formation of a statewide physician provider network. The board also voted to form an individual practice association (IPA). The Garvey Group will first survey state physicians to determine the amount of support for a statewide physician-owned provider network and then explore the market potential of such a network. The Garvey Group was consultant to the Connecticut State Medical Society when it began developing its provider network.

■ **The Oklahoma County Medical Society has** installed its officers for 1994. President Philip Mosca, MD, heads the slate, with President-Elect Michael L. Winzenread, MD; Vice-President Alice E. McInnis, MD; and Secretary-Treasurer J. Christopher Carey, MD. Serving as board members, in addition to these officers, are Edward N. Brandt, Jr., MD; Stephen K. Cagle, MD; Raymond L. Cornelison, MD; Royice B. Everett, MD; Billy J. Matter, MD; Joseph D. Parkhurst, MD; Russell G. Postier, MD; Vadakepat Ramgopal, MD, Richard Vertrees Smith, MD; and Rebecca Goen Tisdal, MD. Ex officio members are Jon C. Axton, MD; Donald R. Carter, MD; William O. Coleman, MD; M. Joe Crosthwait, MD; Sara Reed DePersio, MD; Carol Blackwell Imes, MD; Perry A. Lambird, MD; Douglas W. Voth, MD; and Roland A. Walters, MD.

■ **The Tulsa County Medical Society Board of** Directors for 1994 includes new TCMS President Boyd O. Whitlock, MD; President-Elect David J. Confer, MD; Vice-President W. Frank Phelps, MD; Secretary-Treasurer Edythe P. Harvey, MD; Michael B. Clendenin, MD; William A. Geffen, MD; C. Wallace Hooser, MD; Douglas C. Hubner, MD; John A. Minielly, MD; Kenneth A. Muckala, MD; Richard C. Slagle, MD; and James A. Young, MD. James S. Millar, MD, was elected to a three-year term on the Board of Censors. ❑

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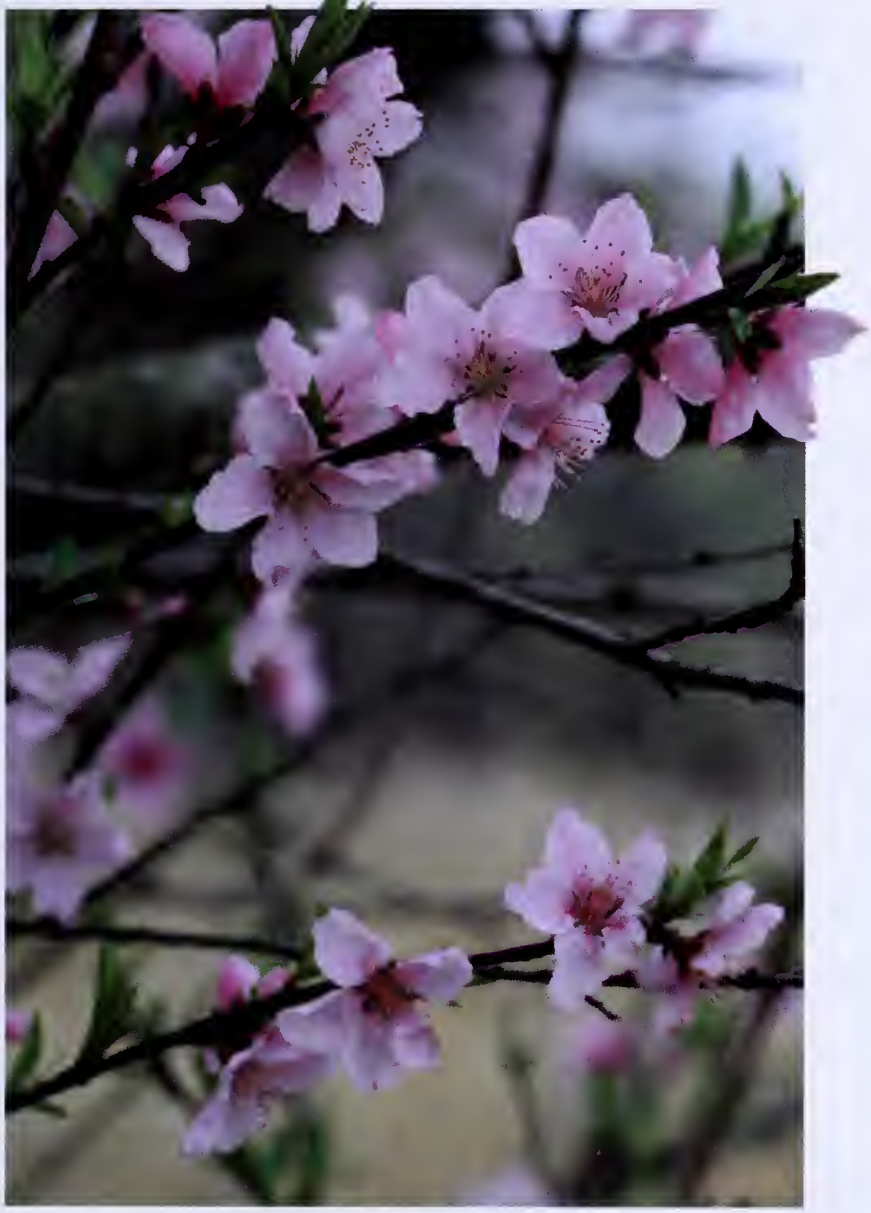

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THE AMA—HEALTH SYSTEM REFORM
Physician Membership and Representation

Perry A. Lambird, MD, MBA

"THE AMA DOESN'T REPRESENT ME." Really?

In traveling throughout Oklahoma, it is clear that two issues are stimulating much conversation among physicians, health system reform and the non-discriminatory nature of AMA membership. Some current AMA policy represents decades of discussion and debate. Perhaps a review of those issues from someone who has actually been there might shed some light on obviously murky misperceptions, for they bear directly upon the reform debate, membership, and physician representation.

HEALTH SYSTEM REFORM

The AMA does not support any of the current bills on health system reform, specifically including Clinton's and Cooper's. It does support the principles developed by the AMA House: Free choice of physician, universal access, physician control of medicine, patient sovereignty, tort reform, administrative simplification, antitrust relief, and market-based approaches to cost containment. The AMA opposes a single payor system a la Canada or Great Britain, budget limits and price controls, bureaucracy, government control of medicine, and interference with the doctor-patient relationship.

AMA policy is clearly attuned to the wishes of most Oklahoma physicians. This is not a surprise since Oklahoma physicians, through their AMA delegation, have been a dominant force in creating that policy.

What seems to be the case is that some physicians, unintentionally or deliberately, are confusing political strategies with policy. The object of being involved with health system reform is to gain an end point as close as possible to the desired policy. To achieve that goal requires on-going communication with all sides in the current national debate. We have those communication channels and through them hope to implement AMA policy.

For illustration purposes, consider the AMA goal of universal access to medical care. This underlies all current reform proposals, but the means vary widely. The largest single congressional block of bill sponsors favors a single payor, government-run system of national health insurance. Although the AMA opposes this approach, it can work in sympathy with the sponsors in achieving a common goal through alternate means. The AMA will support some mixture of employer-provided insurance, individually provided insurance, and/or Medisave Accounts ("health IRAs"). This posture permits mutually supportive discussions with the second largest Congressional group which advocates an employer mandate (a group which includes key committee chairs), and virtually all of the other proposals which envision some form of an individually based system.

What has this approach yielded to date? First, by working with the Clinton administration, many "wonderful ideas" that would have been devastating to American patients and physicians were deleted from the Health Security Act before it was even presented to Congress. Second, by refusing to compromise on price controls, budget limits, and bureaucracy, and by informing virtually all members of Congress what these would mean for patients, we effectively killed the heart of the Clinton plan by the time that it was introduced. Make no mistake, the Clinton plan was dead on arrival. Third, by knitting together the non-single payor groups around various alternatives, we have successfully shifted the focus of the debate to the alternatives!

Never, ever, confuse policy, goals, strategy, and tactics. The AMA has done a brilliant job of representing Oklahoma patients and physicians.

In the months to come, with continuing hard work and clear focus, the AMA is likely to deliver a health system reform package that is absolutely the best achievable in a very difficult political environment.

Could we do more? Of course. If all physicians were AMA members and if all physicians were sustaining members of AMPAC and OMPAC, we would speak with even greater authority and could change the individuals in the Congress and in the legislatures that need to be removed. As long as the average annual physician contribution to OMPAC/AMPAC remains $8, we need to rely on negotiation and wits. Them with the gold rules.

MEMBERSHIP

To be blunt, membership is power.

Within the AMA, and by virtue of our unified membership, we have, and have used, delegation strength to establish reams of AMA policy. We have more delegates than Alabama, Indiana, Iowa, Maryland, Minnesota, Washington, Wisconsin, and many others. We have as many as Missouri, Virginia, and Tennessee. Yet these states are much larger than we. We cannot win all battles. But we have won almost all in which we have been engaged. Oklahoma is elegantly represented.

Nationally, in Congress and before regulatory agencies, membership is also power. Everyone who declines to join weakens our power incrementally. This weakness has been exploited by President Clinton, who has attacked AMA opposition to his bureaucracy-laden plan by stating that the AMA does not represent physicians. It is impossible with our current media to insert the appropriate "yes, buts...." In Oklahoma, for example,

osteopaths are not eligible for membership, but they are always counted as eligibles in political discourse. Only actual members count.

Speaking of membership, my desk, like yours, has been littered lately with letters from the misinformed concerning AMA "endorsement" of homosexual lifestyles. Perhaps some facts might be of interest.

First, the AMA, from personal observation over the last two decades, has never discriminated against anyone. The only effective qualifications for membership are to be an ethical physician or medical student and to pay dues. The AMA House is a complete microcosm of American physicians.

Second, the AMA does not "endorse" any lifestyle. Because of the public health implications of smoking, seat belts, and numerous other issues, it does address "social" issues, often heatedly and with protracted debate.

Third, the debate about amending the bylaws to prohibit discrimination in AMA membership on the basis of sexual orientation was not about endorsing lifestyles but about how best to express the fact that the association has never discriminated in the first place! My personal view was that the time had come to end a "laundry list" approach (race, sex, national origin, etc.), since even our relatively small Board of Trustees includes whites, blacks, men, women, international medical graduates, and so forth. The Council on Constitution and Bylaws, however, had not utilized that approach, so it was not an option for the House. The practical alternative was to spend hours in the House debating what was in fact a non-issue or using the time of the House to debate another topic—AMA policy toward health system reform.

Fourth, all of us tend to forget that we do not like democracy when we are in the minority. The AMA House may well be the most democratic organization that exists in the world today. Every six months it addresses hundreds of issues—each needing only one sponsor. Every member of the association, all 300,000 of us, can speak essentially without limit at any reference committee meeting. The reference committees are charged with preparing business for the House along the lines of the testimony received by them. In the case of the bylaws, the House adopted the recommendation of the reference committee.

Fifth, membership in an organization does carry some responsibilities beyond merely paying dues. The great bulk of the business before each meeting of the AMA is published in synopsis form in *AM News* and is delivered to the preferred mailing address of every member in the weeks preceding the meeting. This gives each member an opportunity to talk with a delegation member concerning any issue, as well as the opportunity to attend the meeting and express his or her own views personally.

In short, members count, members control the AMA, and without AMA membership, American medicine is finished.

REPRESENTATION

It is also useful to consider the alternatives to the AMA for political representation.

A unified voice pays dividends; a divided voice exacts penalties. In Canada, all the provinces but Quebec negotiate fees with the provincial medical society. In Quebec, negotiations are with two different medical groups. Is it a surprise that fees in Quebec are far below those in the other provinces?

Some believe, mistakenly, that the specialty societies could replace the AMA. Look at two illustrations, however.

Which of the following specialty societies should we choose to represent us on health system reform?

1. The American College of Surgeons which has endorsed the single payor system?
2. The American College of Physicians which has endorsed the entire Clinton proposal?
3. The American Society of Internal Medicine or the American Academy of Family Physicians, both of which have endorsed an employer mandate to the exclusion of all other options?

The AMA is faced not only with the difficulty of achieving its policy goals in the face of presidential opposition, but also must contend with the dissonance created by specialty groups pursuing different policy goals but purporting to speak for "physicians." The burden is very heavy.

Second, our specialty organizations are often disingenuously self-serving. I have seen specialty groups trumpet a "win" to their members when, in fact, it was the AMA that served up the victory. I have seen specialty groups decide to "support" 34 items but leave 32 of them to the AMA alone to do the grunt work of getting them adopted. The AMA carries a huge load without effective support while the involved specialty groups devote their energies to a narrow and comfortable issue. Support is withheld when it is due and credit taken when not deserved.

In truth, if we were to dissolve the AMA, we would have to reinstitute it tomorrow. There simply is no alternative.

CONCLUSION

Change is upon us, as physicians, as patients, and as Americans. We have a simple choice. We can increase our OSMA and AMA membership activity and PAC contributions and attempt to serve up an honorable victory. Or we can withdraw our membership, decrease our involvement, and accept an ignominious defeat. The choice is ours. □

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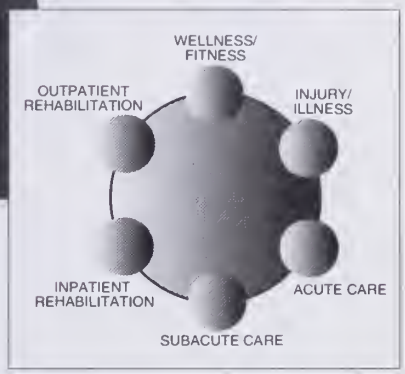

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

National advertising representative: State Medical Journal Advertising Bureau, Inc., 11437 West 106th Street, Overland Park, KS 66214, (913) 888-8781, 1-800-788-2360,

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JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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ABOUT THE COVER

Spring splashes color across a winter-weary state. Photograph by Robert M. Smith, MD. Art direction by Graphic Art Center, Oklahoma City.

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EDITORIAL

Ray V. McIntyre, MD

Still an Awesome Duty

The practice of medicine is a complex intellectual endeavor, perhaps, in some circumstances, the most complicated integration task the human brain ever has to accomplish. Physicians need a long, broad, and deep education, and must take a moral perspective of human needs to their daily work. To ensure that Oklahoma patients are treated by competent physicians, the Oklahoma State Board of Medical Licensure and Supervision issues licenses and license renewals. This awesome duty is regularly discharged by the sanctioning, disciplining, license suspension, or removal of "incompetent, unethical and malfeasant practitioners," even though the diagnosis and conviction of these fallen angels is also a most complex intellectual task.

Somehow, Oklahoma presently finds itself a national leader in the rate of sanctions and license revocations of physicians. For the past several years, Oklahoma has been in the top ten percent, and at least twice has been Number One in the per physician rate of revocations. Regardless of our physician quality, the State of Oklahoma has developed a national reputation for operating a "Medical Gestapo" in physician supervision.

The OSMA should examine this phenomenon and determine if the Medical Practice Act is being administered fairly. The practicing physician must have objective standards to meet that have been devised by impartial practicing physicians. The license to practice medicine should not be jeopardized by the interpretations of lay administrators or by attorneys who have never attended medical school. We take note that the Medical Practice Act and the Rules and Regulations governing the procedures of the Board of Licensure were written by attorneys for use in a judicial adversary system. Board of Licensure activities have no similarity to a physician peer review evaluation, and actual damage to practice privileges may result from *any* action taken by the Board.

A bill (HB 2123) to revise the Medical Practice Act is in the Legislature, and the revision would considerably enlarge the powers of the Board. Noteworthy is the establishment of unspecified continuing medical education as a condition of license renewal. The bill also authorizes a requirement of unlimited community service as a disciplinary measure.

Another feature of the new bill would change the office of Secretary of the Board to a salaried position. Physician service on the Board is thankless public service travail with many unpleasant aspects. We believe the service of all Board members should be compensated with a reasonable per diem payment for days served, paid from funds generated by the fees charged the licensees. Also, serious consideration should be given to reducing the term on the Board to three to five years, with one re-election allowed.

We have some question that the Board is using their present powers equitably enough that they should be granted additional powers. We believe that CME evaluation is far beyond the capacity of the Board to administer fairly. If this bill is passed, it should be amended to include a "Bill of Rights for Physicians."

Ray V. McIntyre, M.D.

President's Page

G. Lance Miller, MD

What's Past Is Prologue

This will be my last communication with you as president of your association. This last year has presented many challenges. Your leadership and staff have kept up a frenetic pace in attempting to meet these challenges. In the beginning we carefully mapped out what we felt would be the key issues and concerns for the year, but as time passed those guidelines were necessarily changed to meet new and pressing challenges. It is my belief that we have taken very aggressive and rational approaches to many of the problems we face and that we have a number of ongoing programs to deal with the challenges.

I have had the pleasure of traveling far and wide to visit many county societies and in doing so have attempted to enlighten our membership about the critical importance of becoming involved in the political process in a meaningful way. Our new IMPACT Program is in its infant stage at this time; in the next few months we hope to start the engine and find out just how effective we've been in convincing state physicians that this program is extremely important not only to themselves but to their patients.

We face many challenges in this new legislative session, including a multitude of paraprofessionals who have introduced bills to establish themselves as gatekeepers with the privilege of writing prescriptions. Of course, health reform remains a major issue at the state level and we will be working intensively with the health care legislators in an attempt to keep sanity in the health system reform laws that are passed. The efforts of our task force on Health Access Oklahoma, which developed a very rational and workable health reform plan, have been warmly received by legislators, and I feel they will go a long way toward establishing the OSMA as a reliable and rational force in shaping health care reform.

As I stated in my very first President's Page, an overriding concern for me is whether our profession can develop the unity necessary to deal with national and state lawmakers during these tumultuous years of determining how our health care system will function in the years to come. As I feared, many of the specialty societies have succumbed to efforts to splinter organized medicine and are putting specialty interests ahead of the interests of the entire profession. It saddens me greatly and I am sure it will weaken organized medicine when it comes to the delicate process of negotiating for health system reform principles. I urge all of us to communicate with the national leaders of our specialty societies and urge them to act professionally and not be a tool for the politicians.

I wish to thank all the physicians who have worked so hard this year to support my term as president and to fulfill the tasks that I set out at the beginning of my term. The officers of the association have taken their responsibilities seriously and have worked like trojans through the many tasks that we have attempted to complete. There are many physicians in this association who are not officers, and not obligated to extend the degree of energy and effort that they have over this year, and to those individuals I am deeply grateful. I am also deeply grateful to the extremely professional and competent executive staff who have never been under such stress nor put so much time into their work.

Lastly, I wish to commend to you your new president, Dr. Jay Gregory, of Muskogee, who is eminently prepared to assume the responsibilities of president and in whom I have a great deal of trust and confidence. I urge all of you to afford Dr. Gregory the same support that you have afforded me, and I personally pledge to Dr. Gregory that I will continue to be at the service of this association whenever needed.

G Lance Miller, MD

Alzheimer's Disease: Pathophysiology and the Hope for Therapy

Roger A. Brumback, MD; Richard W. Leech, MD

The approximately 4.5 million Alzheimer's disease victims in the United States represent a major public health problem resulting in annual expense topping $100 billion, with the Oklahoma share being about $500 million. This problem will be even larger as the baby boomer generation reaches the ages of maximum prevalence of Alzheimer's disease. Research has provided only meager insights into this devastating disease, but has permitted some rational hypotheses concerning potential treatment. Still, the definitive diagnosis of Alzheimer's disease requires pathologic confirmation. In interviews with families following the autopsy, it is apparent that the disease is poorly understood by both the family members and many of the health care workers with which they havedealt. Thus, this review (based upon the questions raised by families and their physicians) examines the currently understood mechanisms and pathogenesis of Alzheimer's disease, the hereditary patterns, and the hope for therapy.

SINCE ITS AUTHORIZATION BY LEGISLATIVE MANDATE in 1990, the Diagnostic Center for Alzheimer's Disease has provided diagnostic confirmation in a large number of cases of neurodegenerative dementia, of which only about 90% are Alzheimer's disease while the remainder represent a variety of neurodegenerative disorders including Pick's disease, Creutzfeldt-Jakob disease, granular cortical atrophy of cerebrovascular disease, and Huntington's disease. The center has also served as a major source of information concerning Alzheimer's disease for physicians, other health care workers, and families from throughout Oklahoma. Their questions repeatedly concern prevalence of the disorder, pathophysiologic basis of dementia, hereditary factors involved, and potential for treatment. This article is based upon the most common inquiries and concerns.

From the Section of Neuropathology, Department of Pathology (RAB, RWL), Diagnostic Center for Alzheimer's Disease (RAB, RWL), Department of Neurology (RAB), and Department of Psychiatry & Behavioral Sciences (RAB), University of Oklahoma College of Medicine, and Laboratory Service, Veterans Affairs Medical Center (RAB), Oklahoma City, Oklahoma.

Direct correspondence to Roger A. Brumback, MD, BMSB 451, OUHSC, 940 Stanton L. Young Boulevard, P.O. Box 26901, Oklahoma City, OK 73190.

Epidemiologic Aspects

Alzheimer's disease is one of the major public health problems in the United States and will become more so in the early part of the twenty-first century as the population ages. Epidemiologic studies[1-4] indicate a prevalence of Alzheimer's disease among individuals over the age of 65 years approaching 10.3%, with an age-adjusted prevalence ranging from 3.0% among individuals ages 65 to 74 years, to 18.7% among those ages 75 to 84 years and 47.2% among individuals ages 85 years and older. Based upon United States Census Bureau data[3,5-7] (Fig. 1), the number of Alzheimer's disease victims now approaches 4.5 million, but will increase to over 9 million by the year 2030 as the baby boomer generation reaches the ages of maximum disease prevalence.

Currently the state of Oklahoma has a population approaching 3.2 million of whom 13% are over

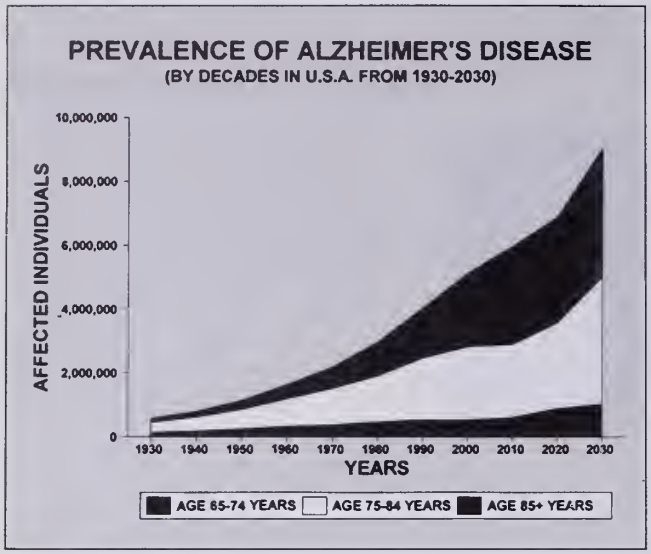

Figure 1. Estimated prevalence of Alzheimer's disease by decades for the century from 1930 to 2030, calculated from United States census data[3,5-7] and epidemiologic studies of Alzheimer's disease.[1-3]

the age of 65 years,[5-7] which means an estimated 20,000 victims of Alzheimer's disease live in Oklahoma. This number will sharply increase over the next 30 to 40 years.

Each Alzheimer's disease victim requires an expenditure of at least $25,000 per year for health care,[7,8] including specialized medical care (50% of the cost), nursing care including nursing homes (35% of the cost), and social services (15% of the cost). Thus, annual expenses for this disease currently top $100 billion in the United States with the Oklahoma share being about $500 million. Of course, this does not include the indirect costs of premature death and the loss of productivity of the victim and caregivers, which may amount to as much as a four-fold greater expense.

Relationship to Normal Aging

Despite the immense suffering and expense resulting from Alzheimer's disease, relatively little research was devoted to this disease until the past ten years[9] (Fig. 2), in part related to the many myths concerning aging intellectual functioning and the aging nervous system. Until the nineteenth century, aging was always equated with increasing wisdom. Although average life expectancy was relatively short (only 47 years by 1910),[10] there were still innumerable examples of individuals surviving and maintaining great productivity into elderly years: for example, Michelangelo was the chief architect of St. Peter's Basilica from ages 72 to 89 years and Goethe completed his opera *Faust* at age 82 years.[7] Yet, European political thinkers of the nineteenth century seized upon scientific reports of pathologic changes in aged brains[11,12] ("with the decay of brain goes a corresponding decay of mind... the natural decline of mental faculties which accompanies the bodily decline of old age"[13]) and enacted into law the concept of "senility" that began at age 65 years and necessitated the end of useful employment along with retirement from normal activities.

During the late nineteenth century, German neuropsychiatrists who devoted great effort toward identifying the pathological substrate of this dementia of aging ("senile dementia")[14] found senile (neuritic) plaques in the cerebral cortex of individuals with senile dementia.[15,16] One of these neuropsychiatrists Alois Alzheimer, working in Kraepelin's laboratory in Munich, presented a paper to the meeting of South-West German Psychiatrists in November 1906 (subsequently published in 1907[17]) describing the case of a woman who at age 51 years developed a progressive dementing illness that lasted 4˚ years. Brain autopsy in this woman who had become demented before the expected age of senility (i.e., "presenile" dementia) showed a large number of neuritic plaques along with a pathologic feature not previously described—numerous neurofibrillary tangles. This presenile dementia (later termed Alzheimer's disease) could be differentiated from the "normal" dementia of senility by age of onset.[18] Subsequent

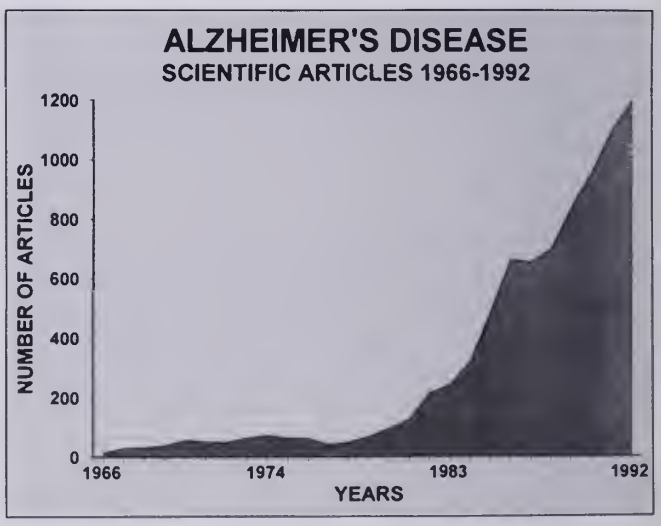

Figure 2. The surge in research interest in Alzheimer's diseaseduring the past decade is evident from this graph of the number of articles on the subject published in scientific journals indexed by *Index Medicus*.

reports finding both neurofibrillary tangles and neuritic plaques in senile dementia served only to confirm the belief that Alzheimer's disease was a form of "precocious senility" due to premature development of the usual aging changes that resulted in senile dementia.[12,19] Not until the mid-1980s did clinician scientists begin to question this notion, abandoning the concept of senile dementia as a normal aging phenomenon and considering Alzheimer's disease a disorder that could begin at any age.[19] Yet, even as late as 1985, when the National Institutes of Health Consensus Conference established histopathologic criteria for the diagnosis of Alzheimer's disease, the idea of "normal aging" changes persisted with the resultant criteria still containing age restrictions (i.e., while the presence of only 2 neuritic plaques per microscopic field were necessary to establish a diagnosis of Alzheimer's disease in 50-year-olds, more than 15 plaques were required for the diagnosis in individuals over age 75 years).[20]

Despite current agreement that the presence of any number of neuritic plaques and neurofibrillary tangles indicates Alzheimer's disease,[21] there is still little understanding of the cause of this disorder. Nonetheless, recent breakthroughs have fostered hypotheses that provide hope for future successful treatment.

Mechanism of Dementia

Dementia is the progressive loss of intellectual/cognitive abilities sufficient to result in impaired social and work performance. Deficits may be identifiable in a variety of areas resulting in memory loss, impaired abstract thinking, decreased problem solving ability, impaired judgment, language disturbance (aphasia), apraxia, agnosia, constructional difficulties, and personality disturbances. These deficits are the result of the loss of the normal interconnections of cerebral neurons. In Alzheimer's disease, the marked reduction in the number of synapses connecting neurons[22,23] is a consequence of the massive death of cerebral cortical neurons.[24] The neuritic plaques and neurofibrillary tangles (which have been considered the pathologic hallmarks of Alzheimer's disease[25]) represent only the tombstones of dead and dying neurons and their neuronal processes (axons and dendrites).

Normally, the full complement of central nervous system neurons is achieved by 24 weeks gestation and the subsequent growth of the brain (that occurs until about age 20 years) entails increases in the number and length of neuronal processes and increased myelination.[26,27] Although during the first few years of life, there is some loss of neurons which do not make proper synaptic connections,[26] approximately a five-fold excess of neurons remains, the extra cells serving a "backup function." Thus, about 80% of nerve cells must be lost in a particular area to result in clinical symptomatology (this is the so called "80% safety margin" for nervous system function). Studies carefully comparing neuronal counts in young (under age 20 years) and old (over age 60 years) individuals have shown up to a 30% loss of neurons associated with normal aging (presumably due to the wear and tear of normal life).[28-31] However, in a disease process such as Alzheimer's disease causing accelerated cell death,[24] clinical symptoms appear when the number of remnant cells is less than the safety margin (that is, when only about 20% of neurons remain viable in areas subserving critical cognitive functions) (Fig. 3). An increasing number of neurologic deficits become evident as nerve cell loss progresses until death ensues from inanition (usually within 5 to 8 years after the onset of clinical symptoms).[9,32]

Based upon findings in Down's syndrome (trisomy chromosome 21), the nerve cell loss in Alzheimer's disease may occur over a period of at least 30 years

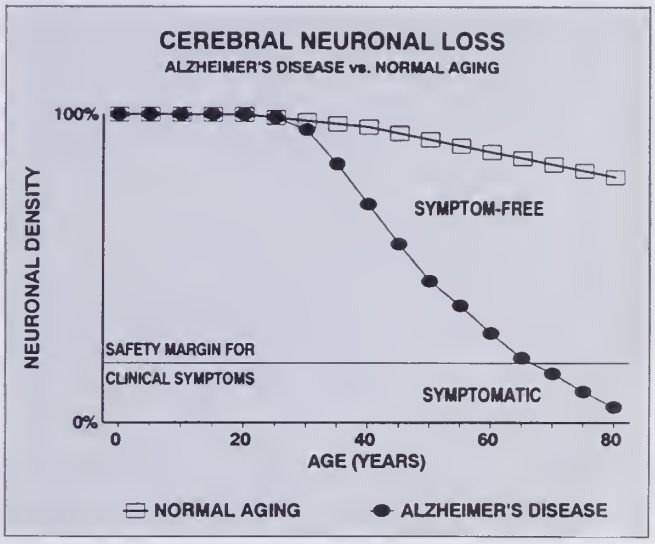

Figure 3. Graph depicting cerebral neuronal loss over the lifetime of an individual. In most people, up to 30% of neurons can be lost due to the wear and tear of normal life, but this loss is far less than the approximately 80% reduction necessary to produce clinical symptomatology (the safety margin for clinical symptoms). However, in Alzheimer's disease, there is progressive destruction of neurons over a 30+-year period such that the number of remaining neurons is less than the 20% necessary for normal clinical function. When too few neurons remain, the patient presents with clinical symptoms.

before clinical symptoms become apparent. Despite being mentally retarded, nearly all individuals with Down's syndrome acquire some cognitive and social skills (for example self-care, sheltered-workshop skills, etc.). However, those who live beyond age 40 years develop a progressive dementia in which they lose those skills, and at autopsy have Alzheimer's disease.[33,34] Careful studies of the brains of Down's syndrome children have demonstrated infrequent neuritic plaques and neurofibrillary tangles indicating that the destructive process of Alzheimer's disease in Down's syndrome patients begins in childhood, although clinical symptoms do not appear until 30 years later.[35] Thus, it can be assumed that elderly non-Down's syndrome individuals developing the clinical symptoms of Alzheimer's disease began to have neuronal destruction 30 or more years previously.

This long prodrome before the onset of clinical symptoms explains in part the increasing prevalence of Alzheimer's disease. If it is assumed that in non-Down's syndrome individuals, the 30+-year destructive process of Alzheimer's disease does not begin until sometime after the brain has reached maximum growth (about age 20 years),[27] an average life expectancy under 50 years (as it was until almost 1920)[10] would mean that most individuals would die during the prodrome and few would live long enough to develop clinical symptoms. On the other hand, with a current life expectancy of 75 years (72 years for men and 78 years for women),[6] many individuals would be expected to live beyond the prodrome and present with clinical dementia from Alzheimer's disease.

The Neuronal Destructive Process

At the current time, it is not exactly clear why nerve cells die in Alzheimer's disease, although a number of hypotheses have been developed based upon biochemical studies of the brains of victims. A great deal of interest has been directed at analysis of the amyloid protein core found in the neuritic plaques.[36,37] Neuritic plaque amyloid protein is different from the amyloid protein found in systemic amyloidosis, and is derived from a protein called amyloid precursor protein (APP) normally found in the nerve terminals.[38] The normal function of amyloid precursor protein is unknown,[39,40] but an aberration in its normal catabolism apparently produces peptide fragments that precipitate to form amyloid.[36,37,41] Some molecular biologists believe that mutations in the amyloid precursor protein molecule trigger abnormal breakdown

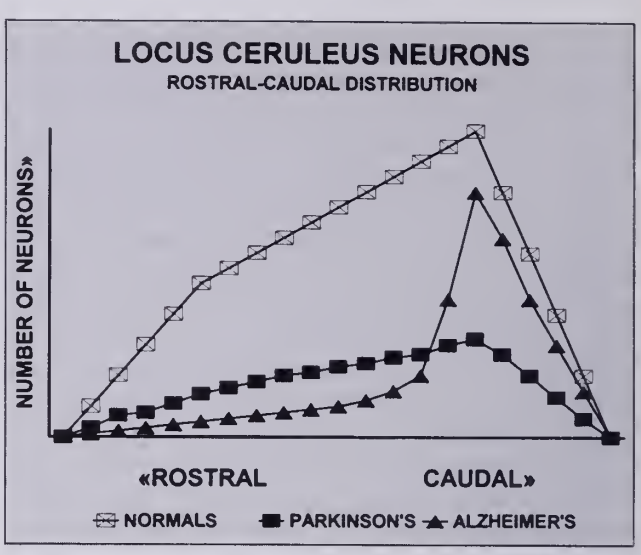

Figure 4. Comparison of neuronal loss in the locus ceruleus in Alzheimer's disease and Parkinson's disease. There is a uniform loss of neurons from the whole rostral-caudal extent of the locus ceruleus in Parkinson's disease due to a metabolic derangement in these catecholaminergic neurons. In contrast, in Alzheimer's disease, the loss involves only those rostral neurons projecting to the cerebral cortex that are normally dependent on neurotrophic factors produced by cerebral cortical neurons, while the caudal neurons projecting to the cerebellum and spinal cord are preserved.

of the protein, with resultant accumulation of amyloid.[42,43] Mutations of catabolic enzymes responsible for amyloid precursor protein turnover could also potentially trigger the abnormal catabolism.

Amyloid may not be the primary cause of the disease, but only a secondary phenomenon of sick neurons which in their agonal condition have disturbed catabolism of amyloid precursor protein.[36,37] Interference with the functioning of neurotrophic factors is one cause for unhealthy neurons. Neurotrophic factors are chemical substances secreted by target postsynaptic cells into synaptic clefts, taken up by the presynaptic terminal, and transported retrogradely along the axon to the cell body where they modify neuronal metabolism (often by affecting protein synthesis).[44]

Experimental blockade of neurotrophic factors results in nerve cell damage or death.[45] One identified neurotrophic factor, nerve growth factor, is normally secreted by hippocampal neurons and retrogradely transported through the fimbria fornix to basal forebrain cholinergic neurons (nucleus basalis of Meynert, the septal nuclei, and nuclei of the diagonal band of Broca).[46] In Alzheimer's disease, interference with the neurotrophic function of nerve growth factor could explain the death of the basal forebrain cholinergic neurons resulting in the

memory disturbance.[47-49] Interference with cortical neurotrophic factors in Alzheimer's disease may also explain the focal neuronal loss in the pontine locus ceruleus, which contrasts with the generalized cell loss in this nucleus in Parkinson's disease.[50] Nerve cell loss in Parkinson's disease results from a metabolic disturbance affecting all catecholaminergic neurons. In contrast, in Alzheimer's disease neurons are only lost from the rostral locus ceruleus where neurons project to the cerebral cortex and are responsive to cerebral cortical neurotrophic factors. Neurons are preserved in the caudal part of the locus ceruleus which projects to the cerebellum and spinal cord.[50] (Fig. 4) A number of additional neurotrophic factors including both peptides (such as brain-derived neurotrophic factor, neurotrophin-3, and ciliary neurotrophic factor) and gangliosides have been identified in the brain.[51,52] Determining which of these or others are responsible for the preservation of certain cell groups and the death of other neuronal populations will require more extensive research.

An intriguing potential clinical correlate for the role of neurotrophic factors is the epidemiologic study of Alzheimer's disease in a relatively genetically homogenous population isolate in the Shanghai region of China.[53,54] This population could be divided into two groups (those with some education and those who were illiterate with no education), with the uneducated group showing a markedly increased prevalence of Alzheimer's disease. Furthermore, those educated individuals afflicted with Alzheimer's disease had onset of clinical symptoms at a much older age. Experimental studies suggest that learning is associated with increased numbers of cortical neuronal synapses and elevated levels of neurotrophic substances.[55] Thus, a longer time was required for the Alzheimer's disease process to interfere sufficiently with the enhanced levels of neurotrophic factors to result in neuronal death.

Hereditary Factors

A number of families with a high incidence of autopsy-proven Alzheimer's disease over several generations have been identified, and the inheritance pattern has suggested an autosomal dominant disorder. Investigators have identified at least three different genetic loci associated with this familial Alzheimer's disease in different families. Mutations have been found in the amyloid precursor protein gene on chromosome 21,[42] a gene on chromosome 19,[56,57] and a gene on chromosome 14.[58] With at least three different genetic loci associated with Alzheimer's disease, it is likely that the disorder is not a simple genetic trait, but instead results from the complicated interaction of genetic load and environmental factors. Thus far only the mutations in the amyloid precursor protein have been characterized,[42] so there is little information about the scope of these genetic influences. With the exception of the possible protective effect of education, even less is known about potential environmental factors.[55]

Potential Therapy

A variety of pharmacotherapeutic measures have been attempted in Alzheimer's disease with varying success. Problems confronting clinical research in this area include the infrequency of early or presymptomatic cases, variable disease progression, and uncertainty about diagnosis (until autopsy). Heterogeneity of the neurodegenerative diseases affecting patients involved in therapeutic studies may be one of the most important stumbling blocks to development of adequate therapy. Studies have repeatedly found that up to one-third [59,60] of carefully screened, clinically diagnosed cases of neurodegenerative dementia will have a disorder other than Alzheimer's disease at autopsy. Thus, even if a potential treatment were beneficial in Alzheimer's disease, the lack of response by a large number of non-Alzheimer's disease victims in a study population could dilute statistical inferences about drug efficacy. Therefore, until other diagnostic markers are discovered, any dementia patient entered into a drug treatment protocol must be followed to autopsy in order to establish the diagnosis and validate the research findings.[8] Of course, this greatly slows therapeutic developments.

Thus far, the basic pharmacotherapeutic approaches that have been used in Alzheimer's disease are: (1) to improve cerebral metabolism and neurotransmission, and (2) to protect neurons from further damage or death.

Improvement of Cerebral Metabolism and Neurotransmission—

Vasodilators. The wide-spread use in the past of cerebral vasodilators was based on the mistaken premise that reduced cerebral blood flow caused Alzheimer's disease. Numerous studies, however, have shown that agents such as dihydroergotamine

mesylate (Hydergine), vincamine, nafronyl, and similar drugs not only do not improve Alzheimer's disease or any other type of neurodegenerative dementia, but also do not significantly increase cerebral blood flow.[51,61]

Psychotropic Agents. Based on another erroneous idea (that altered arousal mechanisms are responsible for dementia), psychostimulants have often been used to treat demented patients.[62] Although a few patients with manic-depression mimicking dementia (so-called depressive pseudodementia) respond favorably, in Alzheimer's disease cognitive abilities deteriorate when the patients are given psychostimulants. Some symptoms of dementia may suggest schizophrenia or psychosis; however, neuroleptics (such as chlorpromazine or haloperidol) given in Alzheimer's disease only exacerbate confusion and memory disturbance or act as a "chemical straightjacket" by inducing significant parkinsonian rigidity and akinesia.[62]

Nootropics. The Belgian pharmaceutical firm UCB coined the term *nootropic* (from the Greek meaning "toward the mind") for a unique class of drugs including piracetam (pyrrolidone acetamide) and pramiracetam.[51,62,63] Through an unknown mechanism of action, nootropics presumably improve brain function and behavior in disease conditions while exhibiting little effect in young or healthy subjects and being devoid of other known pharmacologic properties. In experimental animal studies these drugs prevent amnesia following hypoxia or electroconvulsive stimulation.[63] However, numerous human studies have found no ability of nootropics to improve memory or other cognitive functions.[51,62,64-66] Nonetheless, this has not stopped many "health bars" (particularly in California) from touting mixtures containing nootropic drugs as "smart cocktails" to enhance mental functioning.

Cholinergic Therapy. Following the success of dopamine replacement to ameliorate symptoms of idiopathic Parkinson's disease due to the degeneration of the dopamine-containing neurons of the midbrain substantia nigra, there was great hope that an analogous neurotransmitter augmentation strategy would be successful in Alzheimer's disease. Investigations searching for a single neurotransmitter deficiency in Alzheimer's disease focused on the cholinergic system based upon the cholinergic theory of memory.[67] Administration of anticholinergic drugs (such as scopolamine) which penetrate the blood brain barrier had long been known to produce memory impairment. In contrast, boosting brain acetylcholine levels (by using physostigmine to inhibit acetylcholinesterase-mediated destruction of synaptically released acetylcholine) transiently enhanced memory and learning. Thus, a flurry of interest in treating Alzheimer's disease patients with cholinergic agents followed the discovery of a profound loss of the cholinergic neurons in the nucleus basalis of Meynert and the reports that the degree of cognitive impairment (particularly memory problems) correlated with levels of the enzyme choline acetyltransferase.[68,69] Precursor loading was attempted with both oral choline and lecithin, but neither agent produced clinical improvement or significantly elevated brain choline or acetylcholine levels.[51,66]

On the other hand, intravenous infusions of physostigmine produced an extremely short-lived (minutes only) improvement in cognitive function in some Alzheimer's disease patients.[70] Several synthetic acetylcholinesterase inhibitors with better oral bioavailability and longer duration of action have been developed, the most widely-studied being tetrahydroaminoacridine (THA, tacrine, Cognex) and its hydroxylated derivative HP-029 (velnacrine, Mentane).[71-74] Unfortunately both drugs have significant hepatotoxicity at therapeutic doses. Studies of the few Alzheimer's disease patients able to tolerate the hepatotoxic side effects have generally shown only minimal overall benefit, although occasional patients have a dramatic cognitive improvement. Such a positive response to cholinergic augmentation has stimulated efforts to develop nontoxic agents for use particularly in the initial stages of clinical symptomatology when residual neuronal density is near the threshold (safety margin) necessary for normal clinical function (Fig. 3). At this

The degree and duration of clinical improvement depend on the ability of residual neurons to produce acetylcholine and also on the amount of associated nerve cell loss in other brain areas subserving cognitive functions.

early clinical stage, many nerve cells are only dysfunctional and not yet dead, resulting in the considerable fluctuation in clinical symptoms (from near normal to cognitively impaired) reported by families. At this stage in the disease, inhibition of acetylcholinesterase would increase synaptic concentrations of acetylcholine by preventing its destruction after release from any remaining (healthy or sick) neurons, resulting in markedly improved (possibly normal) cognitive abilities (particularly memory function). The degree and duration of clinical improvement depend on the ability of residual neurons to produce acetylcholine and also on the amount of associated nerve cell loss in other brain areas subserving cognitive functions. Nonetheless, at least for some period of time, such treatment can result in an improved quality of life for the Alzheimer's disease victim.

Ultimately, however, as neuronal death continues, there are too few remaining neurons to produce sufficient acetylcholine to maintain synaptic activity even if enzymatic degradation is prevented. At that point, clinical dementia reappears and clinical fluctuations end. To overcome this dependence on intact neurons producing and releasing acetylcholine, acetylcholine receptor agonists that cross the blood-brain barrier can be used to stimulate directly the post-synaptic acetylcholine receptors. Currently, the only muscarinic receptor agonist available for human studies is RS-86, which produced little clinical improvement in Alzheimer's disease patients at doses that resulted in unacceptable cholinergic side effects (including nausea and excess sweating).[75,76] In some Alzheimer's disease patients, intravenous infusion of nicotine produced memory improvement, but of brief duration due to rapid receptor desensitization.[77] Unfortunately, no synthetic nicotinic agonists are yet available for human testing.

Biogenic Amine and Neuropeptide Agonists. Although biogenic amines have been shown to be reduced in the brains of patients with Alzheimer's disease, there is little evidence that improving aminergic neurotransmission produces any symptomatic improvement.[78,79] This is in contrast to Parkinson's disease, which responds favorably to the administration of aminergic precursors or direct agonists,[80] even though norepinephrine-containing neurons of the locus ceruleus are lost in both disorders.[50,81] Reductions in brain concentration of a variety of neuropeptides has also been reported in Alzheimer's disease, but attempts to alter neuropeptide levels have not yielded any evidence of clinical improvement.[82]

Protecting Neurons from Damage or Death—

As indicated earlier, the health of cholinergic neurons in the basal forebrain nuclei is apparently dependent on the presence of the neurotrophic substance nerve growth factor (NGF) produced by target neurons in the hippocampus and cerebral neocortex.[46-49] Following a variety of experimental brain insults, intraventricular infusion of nerve growth factor preserves the cholinergic basal forebrain neurons while noncholinergic neurons in these same areas degenerate. In contrast, administration of antinerve growth factor antibodies speeds neuronal death.[45] A preliminary therapeutic trial infusing nerve growth factor into the ventricles in Alzheimer's disease patients is underway in Scandinavia, but it will take several years to determine if disease progression has been altered. However, other neurotrophic substances may be just as important in maintaining the health of particular neuronal groups and would need to be included in any therapeutically-successful infusion. Gangliosides (sialic acid-containing glycosphingolipids) have a wide variety of effects including stimulating outgrowth of neuronal processes, inducing axonal regeneration, and protecting neurons from some toxin-induced and hypoxia-induced cell death. Human trials of gangliosides for treatment of stroke and peripheral neuropathies have yielded evidence for clinical improvement, but trials in Alzheimer's disease have not been conclusive.[83]

Summary

Intervention in Alzheimer's disease necessitates a complete understanding of the many aspects of this disorder, which may not be forthcoming for many years. Unfortunately, currently available drugs are associated with unacceptable side effects and produce at best only transient symptomatic improvement. Nonetheless, newer agents are being developed that augment neurotransmission (particularly cholinergic neurotransmission) of remaining neurons and that have a longer duration of action with less toxicity. Such treatment, while not arresting the underlying disease process, will potentially provide an improved quality of life for victims of this dread disease.

Overall, the best hope for long-term treatment of this devastating disease seems to be the efforts directed at developing agents that prevent neuronal

death. Infusion pumps that deliver neurotrophic substances into the ventricular cavities may be an answer, but providing the proper variety and quantity of peptides and gangliosides may be a difficult proposition, although computerization of the infusion systems may be useful. Another means to deliver these substances may be by tissue transplantation into the brain. The beneficial effect of brain tissue transplantations in some patients with Parkinson's disease seems to result not from synaptic transmission by the transplanted tissue but from transplant production of neurotrophic factors that enhance and revitalize remnant nerve cells of the recipient.[84,85] It is possible that appropriate tissue transplantation in Alzheimer's disease could similarly rejuvenate residual neurons, but successful use of tissue transplantation for such effects will require continued research efforts and may be years away from success.

As with many neurodegenerative diseases, progress is slow but data from both clinical and basic science research are stimulating new approaches to therapy. Patients and their families need ongoing and continuous awareness of these differing approaches, and providing them with the information must be part of our responsibility for better and better patient care.

Acknowledgment

The authors wish to acknowledge (1) the Oklahoma State Legislature and the Oklahoma State Department of Health, which provide financial support for the Diagnostic Center for Alzheimer's Disease, and (2) Dr. Fred Silva, Chairman, Department of Pathology, who has promoted the development of this program.

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The Authors

Roger A. Brumback, MD, is professor of pathology (neuropathology) and director of the Diagnostic Center for Alzheimer's Disease at the University of Oklahoma College of Medicine in Oklahoma City.

Richard W. Leech, MD, is a neuropathologist in the Department of Pathology at the University of Oklahoma College of Medicine.

Farm-Related Injury Fatalities in Oklahoma, 1987-1991

Helen Kelley, RN, MN

Farm-related injury is often cited as a major factor in rural health care. This is an attempt to obtain an estimate of the frequency of serious farm injuries in Oklahoma. It is apparent that current available data bases are not adequate to be definitive, but mortality data is available, albeit incomplete. One hundred thirty-two deaths were recorded on death certificates for 1987 through 1991, with falls accounting for nearly half. The other data are summarized, and a review of the farm injury literature is presented. In summary, it is clear that farm injuries are a significant problem and steps to prevent them are required.

While most Americans will acknowledge the value of a healthful lifestyle, many still believe that little opportunity exists to decrease risk of unintentional injury. Rural Americans in particular may be at higher risk for unintentional injury than their urban counterparts. Baker et al[1] compared death rates in five areas selected by population size. Rates were highest in rural remote areas (settlements of less than 2,500 people not adjacent to a metropolitan area). Although many low income people live in rural remote areas, these high death rates were independent of income category.

Many factors may influence these differing death rates. The population itself, the type of injury, the time and distance required to obtain health care, and the quality of the care once obtained will determine much of the outcome of unintentional injury in rural areas.

Direct correspondence to Helen Kelley, RN, MN, 2301 Shady Tree Lane, Edmond, OK 73013.

Although the number of farms and farmers has decreased substantially over the last generation, this occupational group appears to have a disproportionately high number of deaths due to unintentional injury. Indeed, farm workers constituted only 4% of U.S. workers, yet 35% of all machinery-related deaths, 5% of electricity deaths, and 9% of deaths due to falling objects.

Most occupations have experienced lower death rates due to injury over the past several years,[2,3] but the death rate for agriculture remains at 52 per 100,000 workers, five times the combined rate for all other industries.[4] Agriculture fatality statistics generally include the categories of forestry and fishing, but evidence indicates that most of these fatalities are from farming. Agriculture is often a family pursuit, and may be a part-time occupation, therefore involving individuals that are not usually counted as part of the work force. Obtaining the total number of individuals who are at risk for a farm-related injury is a difficult task. In addition, most farms employ fewer than 11 workers, are therefore exempt from Occupational Safety and Health regulations.[5]

Review of the Literature

Several studies have addressed the injury problem in the agricultural workforce. Most analyze the incidence of injury or mortality from encounters with specific types of equipment, or in specific locales. Purschwitz[4] utilized National Safety Council data and fatality data from several states to determine

that fatal farm accidents are most likely to be tractor-related, with half or more of these fatalities due to roll-overs. Substantial numbers of fatalities were caused by other machines, trucks, falls, animals, fire/burns, lightning, tow ropes, poisoning, trees, asphyxiation, drowning, irrigation equipment, and all-terrain vehicles.

Similarly, Etherton et al[6] utilized data from the National Traumatic Occupational Fatality (NTOF) data base to analyze agriculture-related machine fatalities. Here again, a large percentage of fatalities involved tractors, specifically roll-overs, run-overs, and power takeoff incidents. They did not attempt to separate farm-specific incidents, but included logging and manufacturing incidents as well.

May[7] reviewed data from the National Safety Council, as well as several studies that utilized statewide and smaller surveys. He also found that tractors are most often involved in those incidents causing serious injury. Other injuries were related to animals, falls, electricity, and toxic fumes.

Myers[3] utilized data from the NTOF and the National Electronic Injury Surveillance System (NEISS) and found that, in general, injury incidence decreases with age, but mortality rates increase with age. Data in this study did not include workers less than 16 years of age.

Farm-related injuries and mortalities treated over a 12-year period at a Level II trauma center in Wisconsin were analyzed;[8] however, victims dead at the scene or those that died prior to arrival at the referral center were not included. The number of injured patients was 739; injuries involving farm animals and various types of farm machinery were most prevalent. Children were more likely to be injured after falling from a horse, and adults were more likely to be injured in an animal assault; otherwise, the mechanism of injury did not differ significantly according to the age of the injured. Sixteen subjects suffered fatal injuries in this study, with most of those related to incidents with tractors or machines.

Methods

In an attempt to compare the incidence of farm-related injury deaths in the state of Oklahoma with the above results, we analyzed the Oklahoma Vital Statistics data as well as the State Medical Examiner data from 1987 through 1991. Since data are recorded in a different manner in the two data bases, combining the information was not feasible; therefore, each data set was searched for fatalities that appeared most likely to be related to occupational injury on the farm.

The Vital Statistics data base was searched for those deaths listing the decedent as "occupation: farmer." All "agriculture-related but not farm" occupational categories in the Vital Statistics data were eliminated from analysis to identify injuries most likely to occur to farmers. Eleven categories of injury were searched: animal, not otherwise specified; animal ridden/driven; electricity; excessive cold; excessive heat; falls; fires/burns; machine-farm; machine-other; stuck by object; and weather/lightning.

We also attempted to identify what may have been farm fatalities among those unattended deaths included in the State Medical Examiner data base. It was not possible to search according to occupation; instead, the premise or location of the fatality was used to determine those deaths most likely to be farm-related. The locations selected were: pasture/field, ditch, wooded areas, agricultural facility (farm, ranch), and feedyard/feedlot facility. Causes of death examined included: animal/insect, electrocution, exposure, falling object, falls, fire/burn, horse, machine-farm, machine-other, and weather/ lightning.

Results

Table 1 includes data from similar categories in the two data bases. Causes of death have been combined where appropriate to facilitate comparison.

Of the 132 fatalities in the Vital Statistics data, only 6 were female and all 6 were Caucasian; however, they comprised the totality of female farmers listed in all injury categories of the data base. Three

Table 1. Comparison of Selected Categories of Fatalities in the State of Oklahoma, 1987-1991

Vital Statistics			State Medical Examiner		
Category	n	Mean Age	Category	n	Mean Age
Animal	6	49	Animal	10	28
Electricity	3	46	Electrocution	3	36
Exposure	14	70	Exposure	17	52
Falls	60	81	Falls	16	49
Fires/burns	18	76	Fires/burns	10	52
Machine, farm	18	61	Machine, farm	34	56
Machine, other	3	51	Machine, other	5	52
Struck by object	10	65	Falling object	12	51
Total	132			107	

elderly women died after falls, one died from excessive cold, and two middle-aged women died from farm machine injuries. Of the 128 male fatalities in the Vital Statistics data, 8 were Native American, 1 was black, and the other 119 were Caucasian.

In the State Medical Examiner data, 14 women were included, with 4 dying of exposure (2 blacks, 1 Native American, 1 Caucasian), 3 from falls (all Caucasian), and 2 after fires/burns (both Caucasian). The following fatalities were Caucasian: 3 after horse-related injuries, and 2 in farm machine incidents.

Only 26 fatalities were recorded in both data bases. These were: 2 Caucasian females (ages 51, 57) and 24 Caucasian males (age range 17-83 years; mean age 57 years). These deaths included animal (5), exposure (3), farm machine (12), fire/burn (1), fall (1), struck by object (3), and weather/lightning (1). Seven of these deaths had different causes recorded in the two data bases. Some of the differences were minor, related to differences in category definition. Others were more divergent, such as machine, farm/other; animal/fall; and animal/drowning.

Discussion

Although the numbers listed in the various categories of these data bases are similar in most cases, it is apparent that the same cases are not being counted. In the Vital Statistics data, a standard definition is not used, so this classification is determined solely by the person completing the form. It is possible that many farm-related deaths may be missed using this method because people may farm as a secondary occupation. With no standard definition of the occupation; the one-acre "gentleman farmer" might be coded the same as the farmer who tills 2,000 acres, but one would certainly assume markedly different chances for occupational injury between the two individuals. In addition, we postulate that men would probably be coded as farmers more frequently than women, leading to women injured fatally on the farm being overlooked as well.

Unfortunately, farm machine fatalities are not coded specifically, so no comparison could be made with other published data. Notably, the State Medical Examiner data contain almost twice as many farm machine deaths as the Vital Statistics data. Although all these fatalities may not be farmers (and therefore, not included in the Vital Statistics data), one must assume that the fatality was definitely farm-related as the location of injury would indicate.

Obviously, not all injuries in the categories listed are necessarily related to farming as such, but they may provide valuable information to guide further data collection. Children younger than 16 years of age are not included in the Vital Statistics data base, and are often at major risk on farms either when helping operate equipment, or when small children accompany parents in farming operations.

Although the State Medical Examiner data do include young children, they also have limitations. Again, we realize that some farm-related injuries will be missed (those that occur in the home are not included), and some may be included that do not belong (injuries that occurred in a rural area, but had nothing to do with farming). This is a problem common to this type of investigation.[9]

A significant limitation to this study is the focus on fatal injuries only. Since no uniform system for reporting non-fatal farm injuries exists, very limited data are available regarding the precise nature of the problem. Accurate data on the incidence of farm-related injuries would include minor injuries, which may result in many days of work loss and/or physical limitation, as well as substantial economic cost.[10] A farm injury survey[11] indicated that 63% of the farm injuries reported were treated at home. Unfortunately, these investigators did not detail the severity of injuries.

To minimize both the incidence and the severity of injury, research is needed regarding the mechanics of injuries to women and children in particular. Presumably, less force is needed to cause injury to these groups, and so efforts at preventing injury must be designed with these forces in mind. Ergonomically designed equipment with built-in safety features will help minimize or prevent injury.[12,13]

In summary, the need for more specific farm injury data is obvious. Without more specifics regarding the nature of injuries, even the existing data cannot be compared with what little is available nationally. The availability of information from the state of Oklahoma provides a baseline. Future efforts should be directed to more specific efforts at farm injury prevention.

Acknowledgment

The author would like to thank Sue Mackintubee, RN, MPH, and staff at the Injury Control Division, Oklahoma State Department of Health, for their assistance, and Edward N. Brandt, Jr., MD, PhD, for his guidance and support.

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The Author

Helen Kelley, RN, MN, is a clinical research associate at the Oklahoma Foundation for Digestive Research and a part-time doctoral student in epidemiology and biostatistics at the University of Oklahoma.

Upper Gastrointestinal Endoscopy: Its Clinical Use and Safety at a Transplant Center

D.H. Van Thiel, MD; H.I. Wright, MD; Stefano Fagiuoli, MD; J.S. Gavaler, PhD

Upper gastrointestinal endoscopy is a widely practiced clinical procedure that has become routine in even the most remote medical environments. Its application at a liver transplant center represents the use of this particular technology at the opposite extreme of medical practice and its use in a unique and severely ill group of patients.

The following is a description of the author's experience with upper gastrointestinal endoscopy at the University of Pittsburgh's liver transplant center. Despite the severity of illness of the patients seen at this center, documented by the high frequency of patients seen with abnormal prothrombin times and low platelet counts, upper gastrointestinal endoscopy performed either for diagnostic or therapeutic procedures has been found to be safe. The disease processes that affect transplant patients both before and after transplantation are not seen frequently in the general practice of gastroenterology. Thus the endoscopist at a transplant center has to be able to recognize, identify, and treat the unique problems seen in a transplant population.

UPPER GASTROINTESTINAL ENDOSCOPY IS WIDELY utilized as both a diagnostic and therapeutic procedure.[1] Initially, it was used solely for direct visualization of the luminal aspect of the proximal gastrointestinal tract in patients with complaints suggestive of acid peptic disease (esophagitis, gastritis, and gastric and duodenal ulcer) and in cases with acute or chronic gastrointestinal bleeding.[2] When used for such purposes, the procedure has been shown to be very safe and as a result, has been accepted widely by physicians and patients.[3] With increased experience, a broader application of upper gastrointestinal endoscopy has occurred, and it is now routinely used for endoscopic biopsies of the upper gastrointestinal tract and as a therapeutic procedure.[4,5]

The safety of upper gastrointestinal endoscopy, when utilized for these expanded purposes, has not been established as clearly as it has when endoscopy is used solely as a diagnostic procedure. When used for these expanded indications, greater technical skill is required of the endoscopist; the patients are often sicker, with fewer being outpatients, and often the procedures are performed either semi-electively or urgently and in situations where patient control and stability are less than ideal.

The routine use of upper gastrointestinal endoscopy at a liver transplant center both as a diagnostic and a therapeutic procedure incorporates all of the problems experienced with this wider utilization of upper gastrointestinal endoscopy. The following study was performed to define types of endoscopic procedures and the safety of upper gastrointestinal endoscopy in such a setting.

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Oklahoma

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112-4481.

Methods

Case Material—

All upper gastrointestinal endoscopies performed in potential liver transplant recipients and in liver allograft recipients at the University of Pittsburgh Medical Center were reviewed for the years 1989 through 1991. The procedures performed were segregated into those performed for diagnostic and those performed for therapeutic purposes.

At each diagnostic procedure, four endoscopic biopsies of the duodenum and five endoscopic biopsies of the gastric antrum were obtained using large biopsy forceps (4 mm) obtained from the Olympus Corporation (Strongsville, Ohio). If any endoscopic suspicion of an opportunistic infection or tumor was noted in the esophagus, brushings were obtained if varices were present. In the absence of varices, two biopsy specimens were obtained. These tissues were utilized for histologic and virologic purposes (2 biopsies at each site) and to determine the presence of *H. pylori* in patients with advanced liver disease both before and after liver transplantation.

A therapeutic procedure was defined as one in which a specific therapeutic use such as variceal sclerotherapy, injection of a bleeding site, or either electrocautery or a heater probe, was utilized to control or eliminate gastrointestinal bleeding.

Case Material Review—

The experience with upper gastrointestinal endoscopic procedures performed in patients within four different disease categories was tabulated. They were: (1) those with cholestatic liver disease (primary biliary cirrhosis and primary sclerosing cholangitis); (2) those with alcoholic liver disease; (3) those with post-necrotic cirrhosis due to chronic hepatitis B virus (HBV), hepatitis C virus (HCV), or autoimmune or cryptogenic liver disease; and (4) those with either hepatic or biliary malignancy.

For each group the prothrombin time and platelet count at the time of the endoscopic procedure was determined. Moreover, the effectiveness of the therapy when the procedure was performed for a therapeutic purpose and, finally, the safety of the procedure were determined.

Definition of Complications—

A major complication was defined as any one of the following: Gastrointestinal bleeding requiring hospitalization or at least a unit of blood, gastrointestinal perforation, a clinically significant aspiration, excessive sedation that required intubation, cardiac or respiratory arrest, myocardial infarction, or death.

A minor complication was defined as any of the following: prolonged conscious sedation (> 3 hours), transient arrhythmia, or hypoxemia requiring the use of supplemental oxygen that had not been anticipated.

Data Analysis—

All results are reported as mean values ±SEM. The Student's T-test was utilized for statistical analysis.

Results

A total of 1,376 elective diagnostic upper gastrointestinal procedures were performed during the study period. A total of 468 urgent diagnostic or therapeutic procedures were performed during this same period, for a total number of 1,844 procedures (Fig. 1A). The distribution of patients endoscopically examined, defined by their primary hepatic disease, is shown in Figure 1B. The number of patients and the number of procedures differed markedly, as many patients underwent more than one procedure. Essentially, every patient seen for a transplant evaluation had a diagnostic procedure prior to orthotopic liver transplantation (OLTx) candidacy acceptance as part of his or her pre-

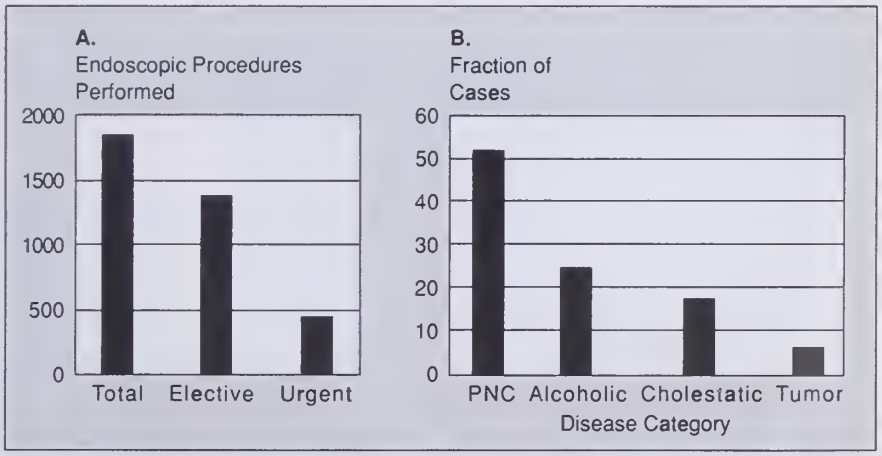

Figure 1. Number of upper gastrointestinal endoscopic procedures performed during the study period on prospective transplant recipients and patients who have had a liver transplant. In 6B, PNC denotes post necrotic cirrhosis.

transplant evaluation. Such procedures identified patients with unsuspected gastric and duodenal ulcers, which were treated medically prior to the stress of the liver transplantation procedure and the obligate use of high-dose glucocorticoids in the post-operative period. Moreover, rare unsuspected gastric carcinomas or esophageal cancers occasionally have been found, removing the candidates with such lesions from further consideration for OLTx. Where gastric ulcers were found, brushings and biopsy samples were always obtained. Occasionally, evidence for cytomegalovirus (CMV) or fungal infection of these ulcers could be documented. In such cases, gancyclovir or nystatin were administered to treat such lesions prior to OLTx. One or more additional diagnostic or therapeutic endoscopic procedures were performed on many of these patients either in the pre- or the perioperative period.

As expected, the prothrombin time and the platelet counts of the patients included in this study were frequently abnormal (Table 1). Patients with post-necrotic cirrhosis and alcohol-induced cirrhosis had the most abnormal values. In contrast, the prothrombin time in the cholestatic group and the platelet count and prothrombin time of patients with tumors often were normal.

The range of therapeutic procedures performed is shown in Figure 2. The vast majority of the therapeutic procedures were performed for control of variceal bleeding. Twenty-one percent underwent emergent endoscopy with sclerotherapy as a result of variceal bleeding. The remaining 79% did not bleed despite a very high prevalence of endoscopically demonstrated grade III/IV esophageal varices.

The complications of sclerotherapy experienced

Table 1. Prothrombin Time and Platelet Counts in Patients Studied

	Protime in seconds x ± SEM (Range)	Platelet Count cells x10³/mm³ x ± SEM (Range)
Cholestatic liver disease	13.9 ± 0.3 (11-20)	160 ± 10 (25-479)
Hepatocellular liver disease	15.2 ± 0.2 (12-31.9)	103 ± 6 (19-418)
ETOH	15.4 ± 0.3 11.7-29.3)	130 ± 7 (25-418)
Tumors	13.9 ± 0.2 (11.2-18.6)	240 ± 18 (45-618)

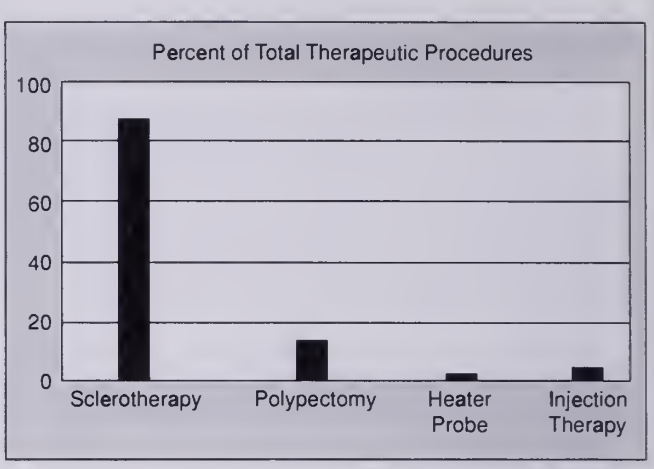

Figure 2. Percent of total therapeutic procedures performed for a given indication shown for either a diagnostic or therapeutic upper gastrointestinal endoscopy.

in elective and emergent sclerotherapy groups are shown in Table 2. Elective endoscopy was performed in those patients with a history of prior bleeding from esophageal varices and who had grade 3 or 4 varices with the additional findings of Wale's lines or hemocystic spots. No true prophylactic sclerotherapy was performed. A significantly higher complication rate was noted for emergent sclerotherapy than for elective sclerotherapy ($p<0.01$). It must be noted that the sclerotherapy performed in the emergently treated patient typically occurs under circumstances of active bleeding with poor visualization, often in obstreperous patients, who are gagging and/or retching despite sedation, and in whom the presence of esophageal motion due to the tachycardia that typically accompanies major gastrointestinal bleeding makes the procedure difficult. The higher complication rate, consisting of strictures, bleeding esophageal ulcers, and perforation in the emergent sclerotherapy group, is explained, at least in part, by these factors and by the aggressive approach used at this center, which consists of high-volume sclerosant administration utilized to eradicate the source of bleeding.

Strictures and bleeding esophageal ulcers, occur-

Table 2. Major Complications of Sclerotherapy

	Elective	Emergent
Esophageal stricture	9%	56%
Bleeding esophageal ulcers	3.1%	10.5%
Esophageal perforation	0.5%	2.9%

ring as a result of sclerotherapy, were treated conservatively, usually with excellent results. Often bouginage was required for esophageal strictures. Sucralfate therapy, used as a slurry, was universally administered for esophageal ulcers. Hemorrhage from esophageal ulcers usually responded to nonsurgical treatment consisting of infusions of octreotide or, in rare cases, vasopressin. However, formal esophagogastrectomy to arrest bleeding was required in two cases following a successful liver transplant.

A rather limited number of procedures were performed for control of bleeding not due to esophageal variceal bleeding. Prior to OLTx, the most common indication for which a specific endoscopic therapy was utilized other than variceal bleeding was Dieulafoy's ulceration, in which equal numbers of cases were treated with either a heater probe/electrocautery or injection therapy.

Following OLTx, the two most frequent causes of upper gastrointestinal bleeding were gastritis and bleeding from a Roux-en-Y biliary-enteric anastomosis. In either instance, no specific endoscopic therapy was possible. In contrast, esophageal or gastric variceal bleeding associated with portal vein thrombosis was a common cause of post-OLTx bleeding for which a specific endoscopic therapy (sclerotherapy) was utilized. Bleeding, however, was not the principal indication for endoscopy following liver transplantation.

The possibility of CMV enteritis was the most common indication for endoscopy post-OLTx. There was no apparent predisposition for CMV infection following liver transplantation according to either the type of liver disease or the therapy for the liver disease prior to transplantation. CMV infection could be demonstrated in 77.3% of patients studied in the second post-OLTx month versus a 9.1% detection rate either before or after the second post-OLTx month.

Table 3. Distribution of CMV Infection in the Upper Gastrointestinal Tract Post-Liver Transplantation

Organ	Percentage
Esophagus alone	0.0
Stomach alone	23.8
Duodenum alone	14.3
Esophagus and Stomach	9.5
Stomach and Duodenum	42.8
Esophagus, stomach, and duodenum	9.5
Total	100.0

Table 4. Prevalence of CMV Infection in Upper Gastrointestinal Tract Pre- and Post-OLTx

	Pre-OLTx	Post-OLTx
Number of patients	88	66
Upper gastrointestinal Tract-CMV	2.2%	31.8%*
Esophageal-CMV	0.0%	6.1%
Gastric-CMV	2.2%	27.2%
Duodenal-CMV	0.0%	22.7%

*$p<0.001$ between pre- and post-OLTx prevalence.

This difference in rates of CMV disease is statistically significant ($p<0.001$). Importantly, the distribution of CMV infection in the upper gastrointestinal tract was not uniform (Tables 3 & 4). Gastric CMV was the most frequent site and was found in 85.7% of the individuals positive for the virus in any organ in the upper gastrointestinal tract accessible by endoscopy (Tables 3 & 4). Among patients awaiting OLTx, CMV when present was found only in the stomach, and then only rarely (Table 4). It was most often recognized as a thickening of the prepyloric and antral folds but also was associated with aphthous ulcers in the same antral location.

CMV infection of the duodenum was most often recognized as focal aphthous ulcers in the bulb and second portion of the duodenum. Other unusual and previously unrecognized findings in pre-transplant patients included cases of sprue; duodenal and antral varices, suggesting thrombosis of the extrahepatic portal vein; and portal hypertensive gastropathy rather than varices as a cause of chronic gastrointestinal bleeding.

Following OLTx, whenever an opportunistic infection was found, the amount of immunosuppression being used was reduced by a third, and specific therapy consisting of nystatin or amphotericin (for tissue invasive disease) or gancyclovir for candida and CMV infections, respectively, was initiated. If antral or greater curvature gastric varices were found, angiography was utilized to assess the patency of the splenic and extrahepatic portal vein.

On two occasions, localized but diffuse petechial hemorrhages were noted in the duodenum and proximal small bowel. In both cases, this finding signaled the identification of an infected arterial graft with marantic embolization of viscera supplied by the graft. In each case, the graft had to be repaired and prolonged antibiotic therapy was required, as is utilized in cases of subacute bacterial endocarditis.

As might be expected, the major complications

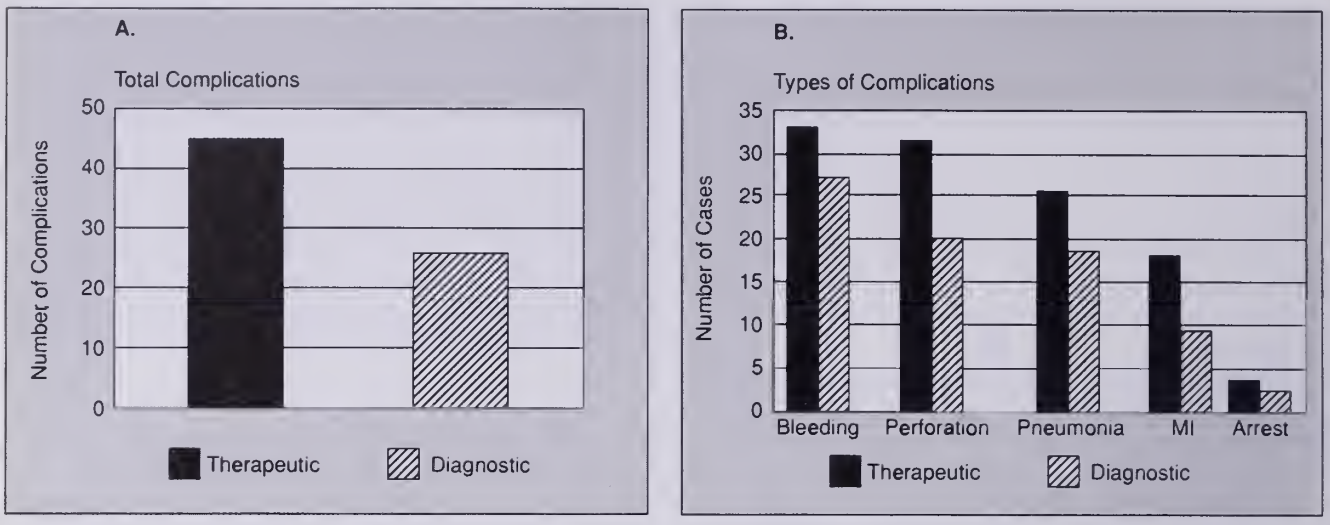

Figure 3. Number (A) and types (B) of complications in percentage of the total occurring as a result of upper gastrointestinal endoscopy performed at a liver transplantation center. The horizontal lines in B demonstrate increments of 5%. Data for therapeutic and diagnostic endoscopic procedures are shown. The number of complications occurring in the therapeutic group is greater than in the diagnostic group, as might be expected.

experienced with diagnostic and therapeutic endoscopy in this high risk population were different (Fig. 3A & B). What was particularly surprising was the rather low number of major complications experienced regardless of the indication for the endoscopy (Fig. 3A). Despite a high incidence of abnormal prothrombin times and low platelet counts (Table 1), clinically important bleeding, even when large endoscopic biopsies were performed, was unusual. Perforations were unusual as well. Two duodenal hematomas occurred as a consequence of endoscopic biopsy. One was in the second portion of the duodenum and produced transient duodenal obstruction which was treated expectantly. The second occurred in the 3rd portion of the duodenum and was complicated by the development of an infected retroperitoneal fluid collection that contributed to the death of this patient after OLTx.

Discussion

There are two major observations to be made as a result of this unique experience. These are, first, that the ability to do upper gastrointestinal endoscopy for both diagnostic and therapeutic purposes is a major clinical component of a liver transplant program. Second, despite the large number of procedures being performed in a particularly high risk group of patients, the frequency of major complications is remarkably small and compares quite favorably with the experience reported for non-transplant patients, most of whom are examined endoscopically as outpatients solely for diagnostic purposes.

The number of upper gastrointestinal procedures performed at a liver transplant center can be staggering. The endoscopists at a transplant center have to be ready to do both diagnostic and therapeutic endoscopy at any time of day or night. More important, they have to be prepared to do these procedures on very ill patients, often with little or no preparation. Worse yet, many of the therapeutic procedures they perform have to be done on patients with abnormal prothrombin times and reduced platelet counts (Table 1); normally these procedures would be used in a routine outpatient setting or in non-transplant cases to identify patients in whom such procedures would be considered dangerous and would therefore be avoided or delayed until such laboratory findings could be corrected.

A third major finding of this study is that, when performed by accomplished endoscopists, a wide range of endoscopy procedures (including sclerotherapy and gastrointestinal biopsies and having both diagnostic and therapeutic indications) can be performed safely in very ill patients awaiting liver transplantation. This suggests that the criteria currently utilized to identify high risk cases in a non-transplant situation may be excessively restrictive.

The two major clinical indications for upper gastrointestinal endoscopy at a liver transplant center are gastrointestinal bleeding and the possibility of an opportunistic infection, usually CMV, involv-

ing the upper gastrointestinal tract. Prior to OLTx, gastrointestinal bleeding occurring as a consequence of either esophageal or, less often, gastric varices, is the most common indication for endoscopy. Despite the fact that potential liver transplant patients are often malnourished and chronically ill, and some are being treated with immunosuppressive agents, opportunistic infections are unusual. After OLTx, both problems occur, but at different times, with bleeding being more likely in the first postoperative month and opportunistic infections being more common in the second postoperative month. Either can occur at later times, but do so only under unusual circumstances of complicated engraftment (technical error) or as a result of over immunosuppression.

As is evident from Table 2, therapeutic procedures that are performed urgently are associated with a much greater rate of endoscopic complications than are those done electively. Many of these complications, particularly the perforations, either delay or transiently prohibit the performance of a liver transplant. In contrast, diagnostic procedures, even those involving gastrointestinal biopsies in patients with abnormal prothrombin times and low platelet counts, can be performed safely and are associated with few, if any, complications (Fig. 3). When complications do occur, however, they can be lethal.

Problems that are seen almost universally in transplant patients but less often in other populations include portal hypertensive gastropathy and duodenopathy, varices of the greater curvature of the stomach and distal esophagus associated with either portal or splenic vein thrombosis, and a host of opportunistic infections ranging from the more common candida esophagitis to CMV enteritis and occasionally fungal infection of an esophageal, gastric, or duodenal ulcer.

In summary, an endoscopist at a transplant center must be experienced and competent in diagnostic as well as therapeutic endoscopy. Moreover, it is important to be aggressive rather than cautious, as any unattended or neglected problem often presents itself later in the transplant recipient's hospital course. Worse yet, when it does, it usually does so in a crisis situation.

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The Authors

David H. Van thiel, MD, is medical director of transplantation at the Oklahoma Transplantation Institution (OTI) and director of the Hepatic and Digestive Disease Center (HDDC) at Baptist Medical Center of Oklahoma in Oklahoma City.

Harlan I. Wright, MD; Stefano Fagiuoli, MD; and Judith S. Gavaler, PhD, are staff members at OTI and HDDC.

Infant Mortality in Oklahoma, 1970-1990

Timothy D. Dye, PhD; Sara Reed DePersio, MD, MPH; Edd D. Rhoades, MD, MPH; Tisha Dowe Webb, MD, MPH; Richard Lorenz, MSPH

Patterns of infant mortality in Oklahoma are unlike the rest of the United States. Oklahoma has a significantly higher post neonatal mortality rate than the United States, with SIDS and congenital disorders being the leading causes of death in that age group. Conversely, Oklahoma has a significantly lower neonatal mortality rate than the United States. Vital statistics information for the period 1970-1990 are examined to describe patterns of infant death in Oklahoma. Despite long-term decreasing trends in infant death, disconcerting trends were observed in 1990 suggesting that a stronger infant mortality reduction strategy is needed in Oklahoma. Potential areas for public health intervention are suggested based on Oklahoma-specific data.

In 1990, 432 INFANTS IN OKLAHOMA DIED BEFORE the age of one year. Oklahoma's infant mortality rate (IMR) in 1990 was 9.1 infant deaths per thousand live births. While variation in the year-to-year IMR is expected, the 1990 IMR represents a 9% increase over the 1989 rate of 8.3 infant deaths per thousand live births. This increase represents the largest proportional increase in the infant mortality rate in Oklahoma in over two decades and follows four consecutive years of decreases in the rate (Fig. 1). Although the 1990 Oklahoma infant mortality rate equalled the infant mortality rate for the entire United States for 1990 (9.1, National Center for Health Statistics 1991), the neonatal mortality rate was nearly 16% lower and the post neonatal mortality rate was almost 26% higher than the U.S. rates. The increase in infant death in Oklahoma and the significantly elevated post neonatal mortality rate represent important public health questions that need to be investigated.

From the Maternal and Infant Health Service, the Child Health and Guidance Service, and Maternal and Child Health Planning and Evaluation, Oklahoma State Department of Health.

Direct correspondence to Timothy D. Dye, PhD, Department of Obstetrics and Gynecology, SUNY College of Medicine, 315 West Tower, 736 Irving Avenue, Syracuse, New York 13210.

These analyses were supported, in part, by the Oklahoma Center for Healthy Infants and Pregnancies Surveillance, funded by Grant Number U50/CCU602873-06 from the Division of Reproductive Health, Centers for Disease Control and Prevention.

Composition of the IMR

The infant mortality rate includes the neonatal mortality rate (the number of infants dying prior to 28 days of age per thousand live births) and the post neonatal mortality rate (the number of infants dying age 28 days to one year per thousand live births). In the United States in 1990, neonatal deaths accounted for 63% of all infant deaths while post neonatal deaths accounted for 37% of all infant deaths. In Oklahoma, however, neonatal deaths accounted for only 53% of all infant deaths, while post neonatal deaths accounted for 47% of all infant deaths.

Examination of the *rates* of neonatal and post neonatal death in Oklahoma and in the United States further illustrate this difference. In 1990, the Oklahoma neonatal mortality rate was 4.8 neonatal deaths per thousand live births, nearly 16% *lower* than the U.S. rate of 5.7 neonatal deaths per thousand live births. The post neonatal mortality rate in Oklahoma for 1990, however, was 4.3 post

neonatal deaths per thousand live births, almost 26% *higher* than the U.S. post neonatal mortality rate of 3.4. Indeed, Oklahoma's post neonatal mortality rate was *significantly higher* ($p<.01$) than the United States' post neonatal rate for 1990, and Oklahoma's neonatal mortality rate was *significantly lower* ($p<.01$) than the United States' neonatal mortality rate.

While the neonatal mortality rate in Oklahoma remained unchanged in 1990 over 1989, and has decreased during six of the prior seven years, the post neonatal mortality rate increased 20% over 1989, the largest such increase since 1972. From these data, therefore, the *increase* in Oklahoma's infant mortality rate for 1990 can largely be explained by the *increase* in the post neonatal mortality rate for that particular year.

Trends in Infant Mortality

It is important to examine Oklahoma's infant mortality statistics in the context of long-term trends in infant mortality rates. Because of the effect that random variation can have on an individual year's rates, it is helpful to examine current rates compared to what rates could be expected based on long-term trend, along with examining a rate's precision.

Overall, the average annual percent decline (AAPD) in the infant mortality rate during the 1970s was 4.9%, with the neonatal mortality rate decreasing at an average of 6.7% per year. In fact, virtually the entire decrease in the infant mortality rate throughout the 1970s was attributable to the dramatic decrease in the neonatal mortality rate during this period. This trend reflected the national trend in neonatal mortality throughout the decade of the 1970s, which saw substantial advances in perinatal technology and practice with subsequently fewer neonatal deaths. In Oklahoma, the post neonatal mortality rate remained stagnant throughout the 1970s, decreasing less than one percent per year.

In the 1980s, the infant mortality rate in Oklahoma continued to decline, albeit at a slower rate (an average of 3.8% per year). Again, as observed in the 1970s, the decrease in the infant mortality rates in the 1980s were largely due to the decreases in the neonatal mortality rates (AAPD = 5% per year). Unlike the decade of the seventies, however, the post neonatal mortality rate decreased throughout the 1980s, at an average of 2% per year. Despite this slight decrease in the 1980s, Oklahoma's post neonatal mortality rate during this twenty-year period was only slight: the post neonatal mortality rate in 1970 was 5.0 post neonatal deaths per 1,000 live births, and the rate twenty years later in 1990 was 4.3. Neonatal mortality, however, decreased significantly during the twenty-year period (the NNMR was 16.1 in 1970 and 4.8 in 1990), although the rate of decline slowed in the 1980s.

Major Causes of Infant Death

The analysis of causes of infant death using vital statistics is compounded by vague, general categories of causes that follow an operational framework rather than a descriptive framework (Dollfus et al 1990:176). Dollfus et al's descriptive framework is used to profile trends in infant death and the composition of infant death in Oklahoma. Furthermore, data is analyzed for Oklahoma using birth cohorts (birth year 1984-1989) rather than death cohorts, meaning that the information presented relates to the year of birth, giving a more precise description of the risk of a particular cohort of infants dying in the neonatal or post neonatal periods. Furthermore, Dollfus et al established a category of infant

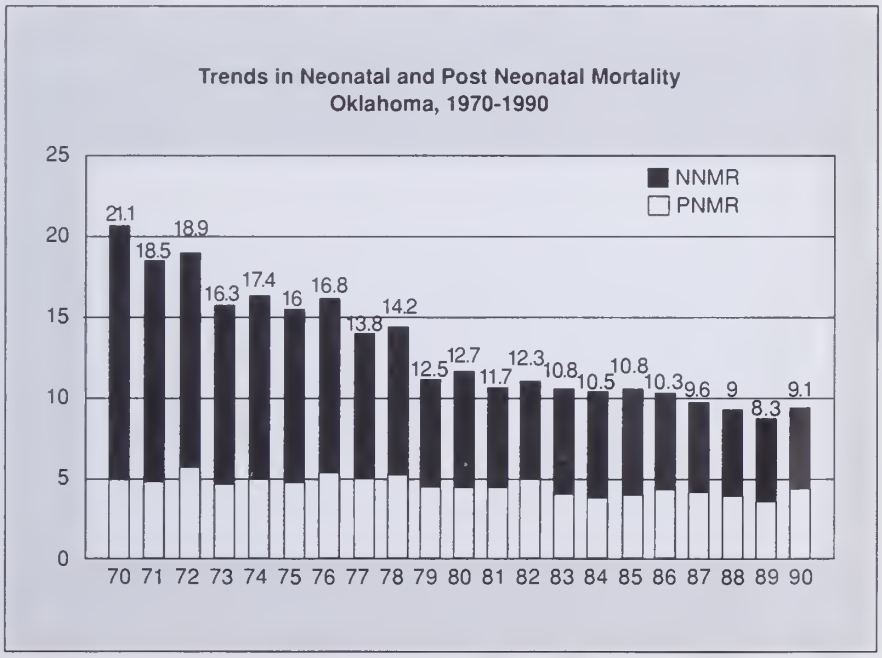

Table 1. Distribution of Descriptive Causes[1] of Infant Death, Oklahoma, 1984-1989 Birth Cohort

Cause	Infant Death n	Infant Death %	Neonatal Death n	Neonatal Death %	Post Neonatal Death n	Post Neonatal Death %
Prematurity and related conditions	811	27.7	745	42.6	66	5.6
Congenital anomalies	684	23.4	530	30.3	154	13.1
Sudden infant death syndrome	535	18.3	32	1.8	503	42.8
Non-perinatal infections	134	4.6	17	1.0	117	10.0
Perinatal infections	116	4.0	106	6.1	10	0.9
External causes and injuries	89	3.0	14	0.8	75	6.4
Obstetric conditions	65	2.2	64	3.7	1	0.1
Birth asphyxia	60	2.1	53	3.0	7	0.6
All other causes	429	14.7	188	10.7	241	20.5
Potentially preventable with prenatal care[2]	1,132	38.7	1,031	58.9	101	8.6
Total infant deaths	2,923	100.0	1,749	100.0	1,174	100.0

[1]Classification scheme using ICD-9 codes, from Dollfus et al. (1990)
[2]Conditions considered potentially preventable with current application of prenatal knowledge, interventions, or technology (Dollfus et al. 1990)

deaths termed "potentially preventable" from a public health perspective. These deaths, in aggregate, are those categories which may have been reduced or avoided given full application of the spectrum of prenatal services that are currently available. (These categories include prematurity and related conditions, CNS anomalies, obstetric conditions, asphyxia, and perinatal infections). Potentially preventable deaths are a broad guide for public health intervention development, although case-specific infant death reviews are needed to detect whether a particular death could be termed preventable.

Major Cause Categories—

Table 1 presents the portions of infant death attributed to specific cause categories for the entire six-year birth cohort, 1984-1989. During this period, prematurity and conditions related to prematurity accounted for the largest portion of infant deaths (27.7%), followed by congenital anomalies (23.4%), and sudden infant death syndrome (SIDS) (18.3%). The remaining infant deaths were caused by perinatal and non-perinatal infections (4.0% and 4.6%, respectively), external causes and injuries (3.0%), obstetric conditions (2.2%), and birth asphyxia (2.1%).

As Table 1 also illustrates, for those infants dying within the first 28 days (neonatal deaths) in birth cohorts 1984 through 1989, the most common cause of death was prematurity and conditions related to prematurity (42.6% of all neonatal deaths). Approximately 30% of all neonatal deaths were caused by congenital anomalies, an additional 6% were caused by perinatal infections, 3.7% were caused by obstetric conditions, and 3% were caused by birth asphyxia. Overall, it is estimated that about 59% of all neonatal deaths (approximately one in three infant deaths) in Oklahoma during the period 1984 through 1989 could potentially have been prevented given full application of current perinatal knowledge and technology.

The major causes of post neonatal mortality, however, vary substantially from the major causes of neonatal mortality (Table 1). About 43% of all infants dying after 28 days but before one year died from SIDS, with an additional 13.1% of all post neonatal deaths due to congenital anomalies. Ten percent of post neonatal deaths in Oklahoma during the period 1984 through 1989 were caused by non-perinatal infections, 6.4% were caused by external causes and injuries, and 5.6% were caused by prematurity and conditions related to prematurity. Overall, only about 9% of all post neonatal deaths in Oklahoma are considered potentially preventable given current prenatal technology, largely because the prevention of SIDS is unclear and the other

Table 2. Infant Mortality Rates, by Region Oklahoma, 1984-1989 Birth Cohort

Region	Infant Mortality Rate	Neonatal Mortality Rate	Post Neonatal Mortality Rate
Metro Oklahoma City	11.2	6.9	4.3
Metro Tulsa	9.9	5.9	4.0
Southeastern Oklahoma	9.6	6.0	3.6
Northwestern Oklahoma	9.3	6.1	3.2
Northeastern Oklahoma	9.2	4.9	4.2
Central Oklahoma	8.8	5.2	3.7
Southwestern Oklahoma	8.2	5.1	3.1
Statewide	9.6	5.7	3.9

Table 3. Infant Deaths by Mother's Age[1]

Maternal Age	IMR (Rate)	NNMR (Rate)	PNMR (Rate)	Cause-Specific Mortality[2] (Rate)			Potentially Preventable (Percent)
				Prematurity	Congenital Anomalies	SIDS	
< 18 years	12.8	7.6	5.2	4.8	1.3	2.4	46.9
18–19 years	12.3	6.8	5.5	4.3	1.3	3.0	43.3
20–34 years	8.1	4.8	3.3	2.3	1.7	1.5	40.2
> 34 years	8.7	5.7	3.0	2.5	2.3	0.8	41.5

[1]Excludes 267 cases from birth year 1984-1989 with no maternal age information; all rates shown are per 1,000 live births, except preventability which is percent of all deaths considered potentially preventable with application of current knowledge and technology
[2]Number of deahts due to indicated cause per 1,000 live births

causes of post neonatal death tend to be less amenable to prevention.

Infant Death by Region—

As shown in Table 2, the major metropolitan areas of Oklahoma (Oklahoma County and Tulsa County) have the highest overall infant mortality rates (11.2 and 9.9, respectively). For the neonatal period, Oklahoma County, the Northwestern Region, and the Southeastern Region have the highest mortality rates (6.9, 6.1, and 6.0, respectively). In addition, Tulsa County has a neonatal mortality rate (5.9) higher than the neonatal mortality rate overall for the state during this same period (5.7). In the post neonatal period, Oklahoma County, the Northeastern Region, and Tulsa County have post neonatal rates (4.3, 4.2, and 4.0, respectively) higher than the state's rate overall for this period (3.9).

Infant Death by Maternal Age—

As shown in Table 3, infants born to teens are at highest risk of both neonatal and post neonatal death, although the proportion of potentially preventable infant deaths with teen mothers is similar to the proportion of potentially preventable infant deaths to older mothers. The elevated neonatal mortality rate among infants born to teens is largely explained by the elevated mortality rate due to prematurity and related conditions among these infants (Table 3). Similarly, the post neonatal mortality rate among infants born to teens is largely explained by the elevated SIDS rate among infants born to teens. Mortality rates due to congenital anomalies are highest among older mothers (age 35 and older).

Infant Death by Race—

As shown in Table 4, a substantial gap exists between the mortality rates of specific race groups. The overall infant mortality rate is highest among infants born to African-American women (14.6), followed by infants born to Native American women (10.1) and white women (9.1). Neonatal and post neonatal mortality rates are also highest among infants born to African-American women (8.7 and 5.9, respectively). The mortality rate due to conditions of prematurity and low birth weight are highest among infants born to African-American women, and the SIDS rate is highest among infants born to Native American women. The mortality rate due to congenital anomalies is similar among infants

Table 4. Infant Deaths by Mother's Race[1]

Maternal Race	IMR (Rate)	NNMR (Rate)	PNMR (Rate)	Cause-Specific Mortality[2] (Rate)			Potentially Preventable (Percent)
				Prematurity	Congenital Anomalies	SIDS	
White	9.1	5.6	3.6	2.5	2.3	1.6	38.0
Black	14.6	8.7	5.9	5.3	2.2	2.5	47.7
Native American	10.1	5.1	5.1	2.1	2.1	2.9	30.3

[1]Child's race from death certificate was used for 267 cases from birth year 1984-1989 with no maternal race information; all rates shown are per 1,000 live births, except preventability which is percent of all deaths considered potentially preventable with application of current knowledge and technology.
[2]Number of deaths due to indicated cause per 1,000 live births.

born to women of all races. Finally, almost 50% of all deaths among infants born to African-American women could potentially be preventable given full application and use of current perinatal technology, knowledge, and interventions.

Infant Death by Birth Weight—

Birth weight is strongly associated with both neonatal and post neonatal mortality in Oklahoma (Table 5). As would be expected, neonatal and post neonatal mortality rates are exceptionally high for infants born below 2,500 gm: in fact, while infants in this weight range comprise 6.6% of the birth population, they comprise 53.7% of the infant death population. For infants considered very low birth weight (<1,500 gm), the major cause of death is prematurity and other conditions related to their birth weight. For infants with birth weight between 1,500 gm and 2,500 gm, the major cause of death is congenital anomalies. SIDS is the leading cause of death for infants with birth weights between 2,500 gm and 4,500 gm, at which point congenital anomalies once again become the leading cause of infant death. Importantly, the prevalence of low birth weight has not decreased substantially in Oklahoma, or in the United States, in several decades. Neonatal mortality has decreased during this same period because of improved survival of low birth weight infants, rather than a decrease in the incidence of low birth weight.

Discussion

Although the Oklahoma infant mortality rate is comparable to the United States' rate, a disconcerting increase in infant mortality was observed in 1990. A large portion of the infant mortality rate in Oklahoma (47%) was attributable to post neonatal deaths, a substantially larger portion than observed for post neonatal death in the United States overall. Indeed, Oklahoma's post neonatal mortality rate is statistically significantly higher than the post neonatal rate for the United States, indicating the post neonatal mortality is a substantial public health concern in Oklahoma. Interestingly, Oklahoma's neonatal mortality rate is statistically significantly lower than the United States' rate, indicating that Oklahoma's infants more frequently survive this critical early period of life. Among certain sub-populations (e.g., African-Americans, teens), however, the neonatal mortality rates remain high.

The infant mortality rate in Oklahoma in the past two decades has, like the rest of the United States, decreased substantially. This decrease is due predominantly to the improved survival of infants in

Table 5. Birth Weight-Specific Mortality Rates and Distribution of Deaths by Case[1]

Birth Weight Group	BW Group as a % of All Births	Neonatal Mortality Rate	Post Neonatal Mortality Rate	Infant Mortality Rate	% Deaths in BW Group Due to Prematurity/Low Birth Weight	% Deaths in BW Group Due to Congenital Anomalies	% Deaths in BW Group Due to SIDS	BW Group as % of All Infant Deaths
Unknown	0.1	152.2	17.3	169.6	46.9	22.4	0.0	1.8
<500	0.1	732.8	8.6	741.4	77.3	2.3	0.0	6.5
500-999	0.4	415.5	64.1	479.6	75.0	4.0	0.9	21.7
1000-1499	0.6	98.2	35.1	133.3	47.8	19.3	6.1	8.6
1500-1999	1.2	34.6	17.3	51.9	19.4	38.7	11.3	7.0
2000-2499	4.3	11.6	9.0	20.6	8.4	38.0	19.4	9.9
2500-2999	15.8	3.2	4.9	8.2	2.8	25.6	31.6	14.5
3000-3499	36.6	1.2	2.5	3.7	4.4	22.1	38.3	15.5
3500-3999	29.8	1.2	2.2	3.3	5.0	14.4	34.6	11.2
4000-4499	9.2	0.7	1.5	2.3	6.3	14.3	30.2	2.4
4500-4999	1.7	0.8	2.5	3.2	5.9	35.3	29.4	0 .6
5000+	0.2	8.1	1.6	9.7	0.0	50.0	0.0	0.2
Total	100.0	5.2	3.6	8.8	30.3	19.0	18.8	100.0

[1]Excludes 267 unmatched cases from birth year 1984-1989, therefore with no available birthweight information.

the neonatal period. Survival in the first month of life is highly dependent on available technology and practices to sustain life at birth, and the rapid development and dissemination of perinatal technologies in the United States in the 1970s, and to some degree the 1980s, is largely responsible for the strong decline in the neonatal mortality rates during this time. The risk profile of women most likely to have infants that will not survive the neonatal period has changed little, and the proportion of women delivering low birth weight infants has remained relatively constant for two decades. Indeed, the proportion of neonatal deaths in Oklahoma that could potentially be prevented using full application and use of current prenatal knowledge, technology, and interventions for the period 1984 through 1989 was almost 60%, indicating that while Oklahoma does not rank poorly regarding neonatal death when compared with the United States overall, a large portion of these deaths could potentially be prevented with full application of risk appropriate obstetric care.

The post neonatal mortality rate, in contrast to the neonatal mortality rate in Oklahoma, remained stagnant throughout the 1970s and decreased only slightly during the 1980s. The preponderance of post neonatal death in Oklahoma is of substantial public health concern. Given current knowledge of prevention of post neonatal mortality, only about 10% of Oklahoma's post neonatal deaths during the period 1984 through 1989 could potentially have been prevented with current perinatal knowledge and technology. The leading cause of post neonatal death in Oklahoma (as elsewhere), Sudden Infant Death Syndrome largely eludes prevention. The large portion of post neonatal deaths in Oklahoma due to SIDS could present state health department officials with the opportunity to develop a state-specific risk assessment program to identify those children at elevated risk of SIDS, and to develop program interventions that target the reduction of SIDS.

Ironically, Oklahoma County, where a large portion of the state's resources and policymakers are located, has consistently higher rates of infant, neonatal, and post neonatal death, indicating an increased need for coordinated, efficient risk assessment and case management programs to reduce infant death in the state's capital. Further research initiatives are needed to explore reasons for this high post neonatal mortality rate in Oklahoma. In addition, research is needed to discover why the neonatal mortality rate is low in Oklahoma when compared with the nation, and if this success could be replicated in other states.

Acknowledgments

The authors wish to acknowledge the cooperation and assistance of the Oklahoma Division of Vital Records. Furthermore, we wish to thank Wansu Chen, Roger Deapen, Don Blose, and Marcia Goff for assistance with tabulations. Patsy Knippenberg provided clerical and graphic support for these analyses.

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Appendix A
Confidence Interval Calculation

An area's infant mortality rate calculated from vital statistics information, while not subject to sampling variability, is subject to random variability (NCHS 1992:50-51). To assess whether point differences between mortality rates reflect true differences (within a specific level of confidence), as this variability may be considerable if the number of events is small, more reliable comparisons of rates are possible by calculating confidence intervals to account for random variation. More detailed explanations of these calculations may be found in the NCHS publication (volume 1, number 2) Statistical Notes for Health Professionals entitled Infant Mortality (Kleinman and Kiely 1991).

The formula for calculating the upper limit of a 95% confidence interval for a mortality rate is:

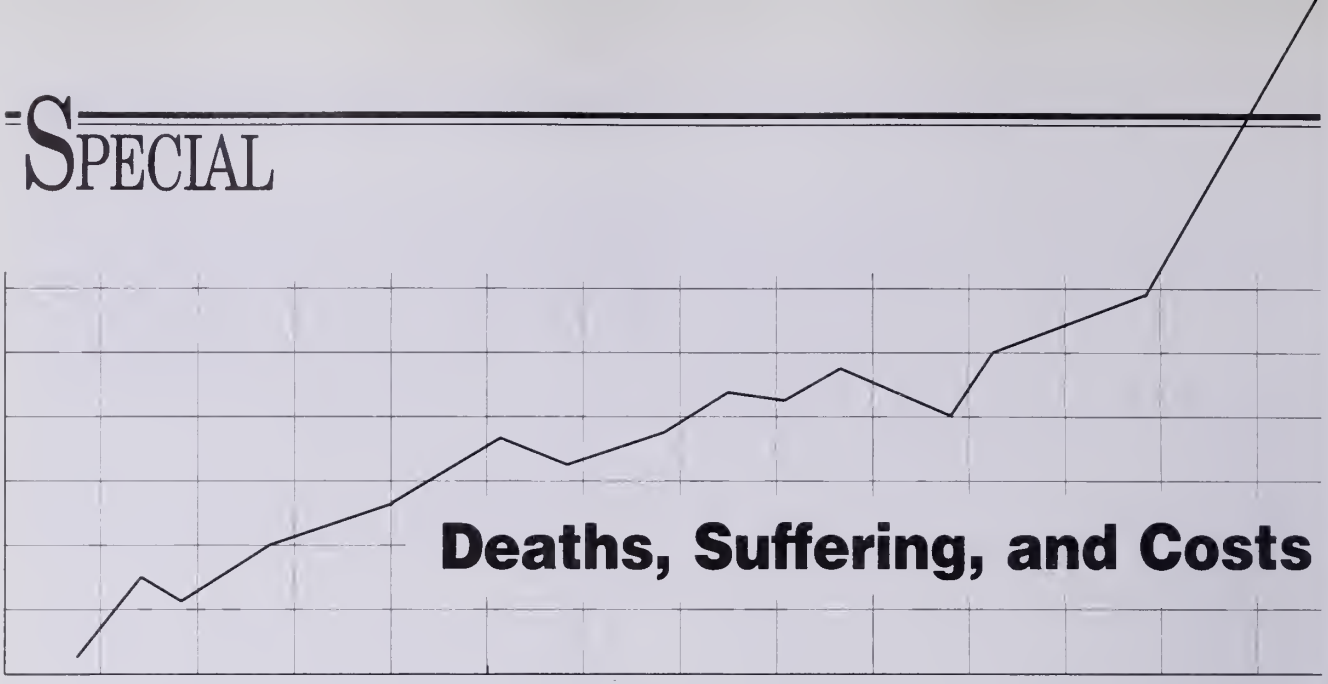
$$r+61.981\sqrt{\frac{r}{n}}$$

where: r = rate per 1,000
n = denominator upon which the rate is based (e.g., number of live births)

Similarly, the formula for calculating the lower limit of a 95% confidence interval for a mortality rate is:

$$r-61.981\sqrt{\frac{r}{n}}$$

SPECIAL



Deaths, Suffering, and Costs

Edward N. Brandt, Jr., MD, PhD

TRAFFIC CRASHES ARE RESPONSIBLE FOR FAR TOO many deaths, injuries, and suffering. Since most of the injuries are major, the costs are also much too high because many, if not most, of these could be prevented by a simple procedure—using a seat belt or child safety seat.

The nation has begun to respond. For example, there were 47,087 deaths in 1988 and five years later, 39,235, a decrease of 16.7%. Yet in Oklahoma, the decrease was only 4.1%! The explanations for the decrease in deaths for the nation are two-fold: a decrease in drunk driving and an increase in seat belt usage. The latter is certainly not true in Oklahoma. Nationally, nearly 63% of drivers used seat belts, while in Oklahoma that figure is only 44% according to the National Highway Traffic Safety Administration. Why? The comparable figure for Texas was 76%. Surely we can beat Texas!

In Oklahoma, from 1988 through 1992, there were 1,618 deaths of vehicle occupants, *1,220 or 75.4% unbelted!* Even more tragically, in children from 0 to 4 years of age, there were 41 deaths, *30 or 73.2% unrestrained.* In children from 5 to 9 years of age, there were 30 deaths *with 23 unrestrained.* What a tragedy that need not have happened!

The story gets worse. In 1992 alone, 3,081 Oklahomans experienced a traumatic brain injury which was fatal or required hospitalization. Of these, 997 or 28% were due to auto crashes. Furthermore, 32 children under 5 years had a traumatic brain injury due to an auto crash. Only 223 of those 997 people were restrained by a seat belt or safety seat. Think of the suffering and costs, most of which were potentially avoidable.

There were 739 Oklahomans who had a spinal cord injury requiring hospitalization in the period 1988 through 1992. Of these, 313, or 48%, resulted from traffic crashes, and 7 were less than 14 years of age. Again, many of these were preventable by the simple act of wearing a seat belt.

Oklahoma physicians are committed to reducing suffering and preventing needless injuries. What can we do about this problem?

First, we can protect ourselves and set an example by always wearing a seat belt.

Second, we can educate our families, our patients, our friends and the public about the importance of wearing seat belts and using safety seats for children.

Third, we can support the resolution passed by the OSMA House of Delegates at its 1993 Annual Meeting to have primary enforcement of seat belt legislation.

Oklahoma has a tragic record of needless deaths, suffering, and medical care costs. We can, and indeed we must, help correct that. Will you help?

Direct correspondence to Edward N. Brandt, Jr., MD, PhD, Colleges of Public Health and Medicine, Room 359, College of Health Building, P.O. Box 26901, Oklahoma City, OK 73190.

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The Author

Edward N. Brandt, Jr., MD, PhD, is professor and co-director of the Center for Health Policy Research and Development at the University of Oklahoma Health Sciences Center in Oklahoma City.

OSMA Annual Meeting to host AMA Pres.-Elect Robert E. McAfee

AMA President-Elect Robert E. McAfee, MD, a surgeon from South Portland, Me., will be a featured speaker at the 88th Annual Meeting of the OSMA House of Delegates next month. He will address a joint luncheon of the OSMA and OSMA Auxiliary on Friday, April 15.

Dr. Robt E. McAfee
AMA Pres.-Elect

The April 15-17 meeting will be held at Shangri-La Resort, near Afton, Okla., in the far northeastern part of the state. Dr. Jay A. Gregory, a Muskogee surgeon, will assume the OSMA presidency at a banquet on Saturday evening, April 16.

The OSMA House of Delegates will hold its Opening Session Friday morning, to be followed immediately by Reference Committee meetings. The Closing Session will be Sunday morning.

The OSMA Hospital Medical Staff Section (HMSS) will also meet Friday morning, and the OSMA Council on Rural Health will meet Saturday morning.

The University of Oklahoma Alumni Association will hold its annual awards dinner on Friday evening.

Golfers should pack their clubs this year, because a tournament is to be held on the nearby Shangri-La Golf Course. For those with other sporting interests, racquetball, tennis (indoor and outdoor), and bowling also are available.

Detailed information on schedules, tickets, and hotel reservations will be mailed to members this month.

Election of AMA Delegates. Elections of AMA delegates and alternate delegates will be held during the Closing Session and this year will be governed by amended bylaws. Any OSMA member wishing to run for a delegate position must have served at least one term as an alternate delegate or have served as an OSMA officer (president, president-elect, immediate past president, vice-president, secretary-treasurer, speaker of the House of Delegates, or chair or vice-chair of the Board of Trustees).

Any active OSMA member may run for an alternate delegate slot, but there must be at least two candidates for each open slot. For example, with three positions to be filled this year, there must be at least six candidates. The OSMA Executive Committee is empowered to solicit candidates for these positions if a sufficient number have not come forward 25 days prior to the election.

Those wishing to run for either delegate or alternate delegate positions must be active members of the association and must provide a letter of nomination signed by a member of the OSMA House of Delegates. The letter must reach the OSMA executive offices not less than 30 days prior to the election.

Dr. Jay A. Gregory
OSMA Pres.-Elect

Both positions are two-year terms that begin immediately upon election, rather than the following January, as was formerly the case. This means newly elected and re-elected delegates an alternates will attend the June 1994 AMA Annual Meeting in Chicago and also the 1994 AMA Interim Meeting in late November or early December.

Incumbent AMA delegates eligible for re-election this year are M. Joe Crosthwait, MD, Oklahoma City; John R. Alexander, MD, Tulsa; and Perry A. Lambird, MD, Oklahoma City.

Incumbent alternate delegates eligible for re-election are Mary Anne McCaffree, MD, Oklahoma City; Boyd O. Whitlock, MD, Tulsa; and David M. Selby, MD, Enid.

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Loss prevention seminars continue; is this your year to attend?

The Physicians Liability Insurance Company (PLICO) is well into its 1994 schedule of loss prevention seminars. Oklahoma physicians, in order to maintain their PLICO coverage, are required to attend a loss prevention seminar every three years. Any insured physician who has not attended such a seminar since 1991 is required to attend one in 1994. Attendance is also mandatory for any physician's assistant, RN, or LPN who is specifically named as a covered employee in a physician's professional liability policy.

Physicians who must attend a seminar this year were notified by mail in late January.

The following is the seminar schedule for the remainder of 1994:

Tuesday March 15	Norman McClain County Med. Soc. OU Student Union	6:30-9:30 PM
Tuesday MARCH 29	Tulsa MARRIOTT SOUTHERN HILLS 1902 E. 71st Street, S.	6-9 PM
Thursday March 31	Oklahoma City Clarion Hotel (formerly Lincoln Plaza) 4345 Lincoln Blvd.	6-9 PM
Sunday April 10	Tulsa State Psychiatric Assn. Doubletree Warren Place 6100 S. Yale (Call Claudine Channell 329-1100 for reservations)	12:30-3:30 PM
Saturday April 16	Grand Lake State Surgical Assn. Shangri-La Resort "Breast Cancer" (OSMA Annual Meeting)	8:30-11:30 AM
Tuesday April 19	Shawnee Pottawatomie Cnty. Med. Soc. Shawnee Country Club	7-10 PM
Thursday April 21	Elk City Holiday Inn I-40/Hwy 6	6-9 PM
Thursday May 5	Enid Best Western 2818 S. Van Buren	6-9 PM
Monday May 9	Chickasha Grady Memorial Hosp. Hospital Medical Staff 2220 Iowa Avenue	7-10 PM
Thursday MAY 26	Lawton HOWARD JOHNSONS 1125 E. Gore Blvd.	6-9 PM
Thursday June 2	Woodward N.W. Inn Hwy 270 South	6-9 PM
Thursday June 16	Oklahoma City Holiday Inn West 801 S. Meridan	6-9 PM
Thursday June 23	McAlester Holiday Inn US Hwy 69 Bypass South	6-9 PM
Friday June 24	Tulsa Tulsa Medical School (for residents) 2808 S. Sheridan Road	8-11 PM
Monday Sept. 26	Muskogee E. Central County Med. Soc. Ramada Inn 800 S. 32nd	6-9 PM
Tuesday Sept. 27	Oklahoma City Clarion Hotel (formerly Lincoln Plaza) 4345 Lincoln Blvd.	6-9 PM
Thursday Sept. 29	Tulsa Tulsa OU College Auditorium 2808 S. Sheridan (OSMA sponsored - all MDs)	6-9 PM

HEALTH DEPARTMENT

Regulations apply to screening for metabolic disorders in the newborn

The Oklahoma Newborn Metabolic Disorder Screening Regulations, revised in 1992, have been developed based on recommendations from the American Academy of Pediatrics (AAP). The regulations designate the physician with responsibility for (a) ensuring that all infants in his/her care have been screened, (b) follow-up of unsatisfactory specimens and abnormal screening results, and (c) rescreening of infants when medically indicated.

The Oklahoma State Department of Health (OSDH) initiated a statewide program for newborn metabolic disorder screening with the passage of the Newborn Metabolic Disorder Screening law in 1965. The screening program began with screening for phenylketonuria; screening for congenital hypothyroidism was added in 1970, and screening for galactosemia and sickle cell diseases was added in

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Health Department (continued)

1991. The most current statement by the AAP was published in February of 1992 and makes further recommendations related to practice issues:

1. In order to ensure all newborns are screened for metabolic disorders, all newborns should be screened prior to discharge from the hospital.
2. All newborns screened prior to 24 hours of age should be rescreened at three to five days of age because a small percentage of infants with phenylketonuria, congenital hypothyroidism, galactosemia, and sickle cell disease might be missed by the early screen.
3. All newborns should be screened prior to transfusion or dialysis if possible since post transfusion/dialysis results may be unreliable, especially for sickle cell disease and galactosemia. If an infant is screened following transfusion or dialysis, a repeat specimen should be obtained at 120 days post transfusion for sickle cell disease and approximately one week post transfusion for the other disorders.

The Newborn Metabolic Disorder Screening Program is an integrated program for detection and management of infants affected by any of four disorders in the screening panel. The OSDH Public Health Laboratory, Maternal and Infant Health Newborn Metabolic Disorder Screening Program, the hospital, and the physician each play a critical role in the success of this program. Questions regarding Newborn Metabolic Disorder Screening should be directed to Garry McKee, PhD, (laboratory), 405-271-5070 or Shari Kinney, RN, MS, (follow-up), 405-271-6617.

From the University of Oklahoma Press

Book examines right to die

Letting Go: Death, Dying, and the Law is the title of a book soon to be released by the University of Oklahoma Press. In it author Melvin I. Urofsky analyzes the pros and cons of withdrawing life support.

Urofsky, a professor of history and constitutional law at Virginia Commonwealth University, examines such landmark cases as those of Nancy Cruzan, Karen Ann Quinlan, and Baby Jane Doe. In discussing the "right to die" issue, however, he widens his scope to include waived stays of execution, "living wills," suicide as a rational option, capital punishment, and differing religious attitudes toward death.

The 224-page paperback is scheduled for publication in April 1994. Price is to be $10.95.

Study: Are emergency room physicians stingy with analgesics?

Physicians do not use adequate analgesia for emergency room patients with acutely painful conditions, according to a study by Lawrence M. Lewis, MD; Laura C. Lasater, MD; and Christopher B. Brooks, MD; of St. Louis University School of Medicine.

An article regarding the study, entitled "Are Emergency Physicians Too Stingy with Analgesics?" was published in the January 1994 issue of *Southern Medical Journal.* According to the article, the study of 401 cases over a 17-day period in eight metropolitan hospitals in the St. Louis area found only one-third of the patients treated in emergency rooms for fractures were given analgesics.

The authors offer the following explanations, discussed in detail, for these physician's reluctance to use analgesics: (1) patient refusal, (2) overriding concern about making the diagnosis, (3) potential to mask more serious, occult injuries, (4) adverse side effects of the medication, (5) potential for drug dependency, and (6) miscommunication (or disagreement) between emergency physician and various consulting physicians.

The study also shows no significant difference in the dispensing of analgesics in reference to fracture site, emergency department setting, or patient age. The authors conclude that inadequate use of analgesics is widespread and that efforts to improve the appropriate use of analgesics in the emergency department are warranted.

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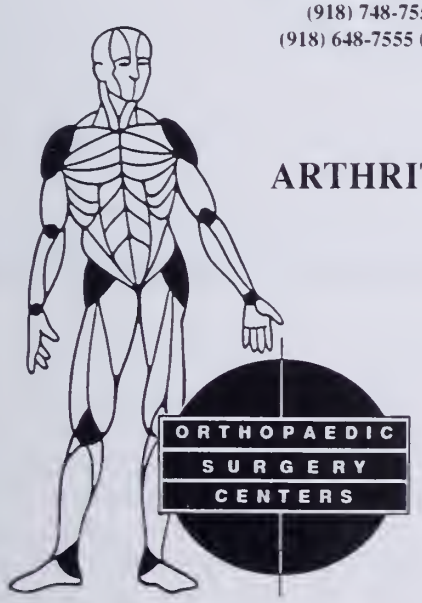

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Restoring the image of medicine: Marcus Welby, where are you?

Marcus Welby, MD, that 1970s television icon, represented the golden age of American medicine. Unfortunately for doctors, Marcus Welby's practice environment—with its virtually unlimited physician authority, autonomy, and prerogative—has disappeared forever. But can the large measure of public trust and admiration the fictional character enjoyed be recaptured by his real-life counterparts? The January issue of *The Internist: Health Policy in Practice,* the magazine of the American Society of Internal Medicine (ASIM), explores the changing public perception of physicians and the medical profession, focusing on what medicine can do to restore its somewhat tarnished image.

In one recent survey, 69% of those polled said doctors are too interested in making money. In another, 35% blamed the medical profession for the nation's health care crisis. Why all the hostility? Part of the problem, says Victor Cohn, one of the nation's leading science reporters and the former science editor of *The Washington Post,* is a kind of crisis of expectations. "We retain an image... of the old, warm family doctor holding patients' hands and dispensing pink pills and homilies," writes Cohn. "Instead, we encounter the modern MD-technologist ordering lab tests and MRIs that warm no hands but get better results."

Cohn emphasizes that, more than time, attention, and expertise, patients want "a feeling that our doctors care about us." Individual physicians need to make patients feel the person behind the stethoscope is interested and concerned about them.

Carol Mullinax, an associate executive director as well as communications director of the Ohio State Medical Association, agrees. Mullinax writes: "The physician who feels this is bunk hasn't heard the amazement—and pleasure—in the voice of a patient who reports, 'My doctor called me at home to see if I was feeling better.'" Mullinax says reviving the Marcus Welby mystique can be achieved if doctors take a customer-service approach to medicine, identifying and correcting problem areas such how long the patient has to wait to see the physician.

William L. Kissick, MD, professor of public health and preventive medicine at the University of Pennsylvania School of Medicine, says the image of the medical profession will be continually shaped by evolving health policy and changes in the practice environment, adding that the public relations implications can be positive. The push for increased cost-effectiveness, for example, could cast patients in the role of collaborators in their own health—and doctors in the part of revered health "teachers."

On a related note, changes in the health care system form the basis of "InnerView." John M. Eisenberg, MD, head of the Physician Payment Review Commission, discusses the implications of health system reform for internists in training as well as internists in practice. Dr. Eisenberg says medical schools need to adopt a new kind of curriculum, one that teaches doctors how to consider "the patient's resources, the cost of care, and the patient's own value system as well as the traditional measures of outcome."

In "In Focus," ASIM Executive Vice President Alan R. Nelson, MD, takes a look at "merger mania," the buying up of medium- and small-sized group practices by hospitals and managed care entities. There's nothing wrong with the trend, he says, as long as the legitimate needs of patients, physicians, and the community aren't swept aside by the new owners.

Building Bridges – Leading the Way

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The 1993 update edition of *Physician Marketplace Statistics.* Provides the latest statistics, at the most

CAPSULES (CONTINUED)

detailed level possible, for answering questions about the practice and practice environment of today's physician. $175 for members, $295 for nonmembers. Call 1-800-621-8335 and request item OP193193LD.

"The Campaign for Tort Reform" is the AMA's newest Video Journal. The 14-minute video and informational packet features leading physicians and experts discussing the problems physicians face due to the inability of the U.S. tort system to resolve medical liability claims in a rational, timely, and cost-effective manner. $13 for members, $30 for nonmembers. Call 1-800-621-8335 and request item OP111993CU.

■ **AMA members can now receive discounts on** a wide range of Xerox products through a new program co-sponsored by Xerox Corp. and AMA Services, Inc. Members are eligible for discount pricing on the purchase of Xerox copiers, facsimile machines, printers, electronic publishing systems, and other document equipment, realizing up to a 30% savings over list prices. For more information, call 1-800-422-0077.

■ **The AMA announced its health system reform** priorities for 1994 during a January 24 press briefing in Washington, D.C. AMA Board Chair Lonnie R. Bristow, MD, presented the following priorities to reporters: (1) Universal coverage; (2) Assurance that physicians, as advocates for their patients, play a significant role in deciding how health care is delivered and financed; (3) Guarantees that all patients have a choice of health plans and physicians; (4) Reforms in the tort system, like those in California; (5) Relief from antitrust laws that prohibit physicians from negotiating quality of care and conditions of patient coverage with insurance and hospital plans; and (6) Creation of new public/private partnership to set and monitor national quality standards.

■ **The National Rural Health Association will** hold its 17th annual national conference, "Building Bridges—Leading the Way," May 25-28 at the Hyatt Regency Hotel in San Francisco. The conference will focus on how rural health care organizations, facilities, and providers can work together in building alliances, networks, and coalitions. It will also explore how these bridges benefit the providers and health workers as well as those receiving care. For registration information, call the NRHA National Service Center, 816-756-3140.

IN MEMORIAM

1993

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William Niebuhr Weaver, MD	March 20
Willie George Long, MD	March 22
William Finis Ewing, Jr., MD	March 31
John Robert McInnis, MD	March 31
Chester Randall Seba, MD	April 4
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John Raymond Stacy, MD	July 6
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John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13

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LETTERS

Wants vs Needs

To the Editor: I certainly enjoyed your editorial "Whence Maturity" [Jan. 94 JOURNAL]. It is nice to have an editorial which is thought-provoking and applies not only to the practice of medicine, but to life in general. I agree with you that most mature individuals do become able to delay the gratification of instinctive needs to an appropriate time and place. However, you will make a real contribution to our society if you can come up with a suggestion as to how most people can satisfy their needs in a timely fashion, without wasting energy on unnecessary wants. Thanks again for your thoughts.

—David Harper, MD
Tulsa

DEATHS

Austin Walsh Webb Haddox, MD
1923 - 1994

Retired Antlers physician Austin W. Haddox, MD, died January 13, 1994. Dr. Haddox, a native of Pawnee, Okla., and 1951 graduate of Washington University School of Medicine, St. Louis, maintained a general practitioner in Antlers for almost 40 years. During World War II he served on active duty for 30 months, 23 of them overseas. He retired from the practice of medicine in 1989 and was awarded OSMA Life Membership status the following year.

CLASSIFIEDS

Classified ads are 50 cents a word, with a minimum of $25 per ad. A word is one or more characters bounded by spaces. Box numbers will be assigned upon request and will add 6 words to the total. Payment must accompany all submissions. **Orders will NOT be accepted via telephone or fax.** Mail ad with payment to OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118. Deadline is the first of the month preceding the month of publication.

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Mammogram interpretations. Nineteen years experience. Board certified radiologist (retired) seeks part-time or possible full-time position. Central Oklahoma preferred. Write Box 46, c/o OSMA Journal.

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Medical Marriages: Balancing Commitments to Family and Profession featuring Dr. Roy & Bev Menninger. July 24-29. Crested Butte, CO. CE Credit: 24 hours. Contact: Menninger Continuing Education, 800/288-7377.

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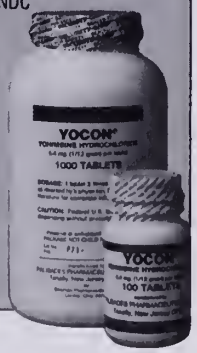

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3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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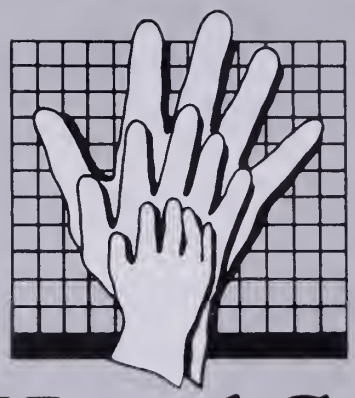

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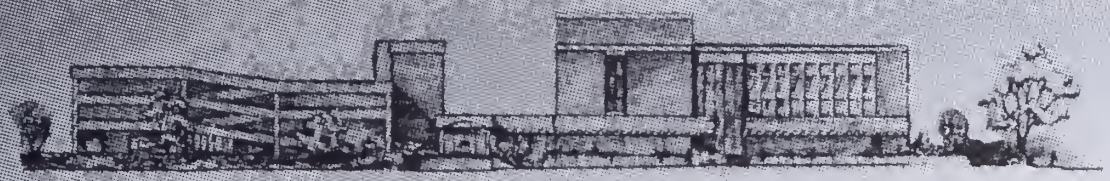

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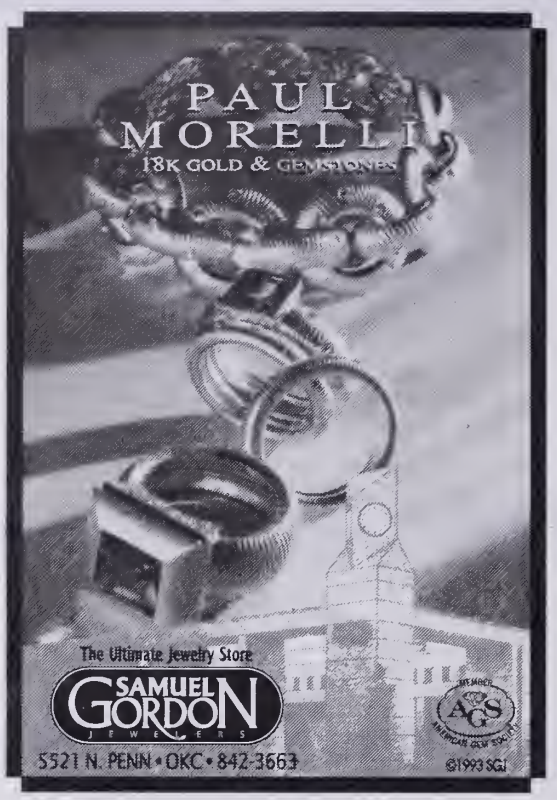

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All manuscripts should adhere to the style adopted by the American Medical Association as illustrated in *JAMA* and detailed in the AMA's *Manual of Style*. An abstract of 150 words or less should accompany each paper and should state the exact question considered, the key points of methodology and success of execution, the key findings, and the conclusions directly supported by these findings. Footnotes, bibliographies, and legends for illustrations should be on separate sheets. References are to be listed in the order of their appearance in the article.

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Authors will receive reprint order forms from the Transcript Press, PO Box 6440, Norman, OK 73070-6440, with their galleys. Other requests for reprints must be made to the Transcript Press within 30 days after publication.

OSMAA State Convention

The OSMAA Annual Meeting will be held April 14-17 at Shangri-La Resort, located on the beautiful Grand Lake O' the Cherokees. The convention officially begins with the pre-convention board meeting scheduled for 3 PM on Thursday, April 14. Following the meeting, a reception will be held to greet our special guests, Darlene Young, secretary of AMAA, and Mildred Taylor, president of Southern Medical Auxiliary. Dinner is planned at a nearby restaurant, The Roadhouse, and is optional.

Friday morning will begin with a breakfast honoring our past state presidents, a breakfast for county presidents and presidents-elect, and a continental breakfast for delegates. The House of Delegates meeting will begin at 9 AM. Officers for the 1994-95 auxiliary year will be installed. At noon we will join the OSMA at a luncheon with speaker Dr. Robert E. McAfee, president-elect of the AMA. After the luncheon, we will take a trip back in time and visit Har-Ber Village, which has been offering visitors a glimpse of the past for 25 years. The 13-acre park includes over 60 buildings which house more than 100 displays of American antiques and memorabilia; most are authentic to the frontier period. Har-Ber Village has been named the state's number one tourist attraction. Other afternoon activity options include tennis, golf, or shopping.

On Friday night the auxiliary will again join the spouses for a casual evening of dining and dancing for an AMA-ERF benefit. Theme baskets from every county will be silently auctioned, with proceeds going to the medical school of your choice.

The auxiliary adjourns the business portion of the convention on Saturday morning after a post-convention meeting of the newly installed board. The OSMA and OSMAA Presidents' Reception and Banquet will be held Saturday night. A special program with entertainment with guest speaker, awards, music, and dancing will put the finishing touches on the 86th year of medical auxiliary in Oklahoma.

—Patricia S. Bass
State Convention Chair, 1993-94

The Last Word

■ **The Oklahoma State Medical Association's** Council on Member Services will sponsor three seminars next month on coding for insurance and billing. The seminars are full day programs and will review the basics of CPT and ICD-9 procedural coding for 1994. The dates and locations of the seminars are April 1, Tulsa; April 5, Oklahoma City; and April 7, Lawton. Specials flyers with additional details and registration information are being mailed to OSMA members.

■ **The Oklahoma Poison Control Center faces** extinction if the Oklahoma Legislature does not appropriate funds for it this year. The center operates from Children's Hospital in Oklahoma City and is professionally staffed 24 hours a day. Emergency funds were appropriated for it last year, at the eleventh hour, but the crisis was only temporarily averted. Norman Representative Laura Boyd, an advocate for the center, is asking physicians to send letters of support for the center to their legislators. Such letters would be particularly meaningful if they relate instances were the center has made a difference in a case. Correspondence should be directed to the House of Representatives, State Capitol Building, Oklahoma City, OK 73105, and the Oklahoma State Senate, at the same address.

■ **Officers and staff members of the OSMA stand** ready to meet with member groups at any time to discuss the status of OSMA's managed care plans. Hospital staffs, specialty societies, county societies, or alliance groups can call OSMA (405-843-9571 or 1-800-522-9452) to schedule a speaker.

■ **Physicians are being urged to talk with their** patients about health system reform. To encourage that dialogue, the OSMA Council on Professional and Public Relations has produced three pamphlets for display and distribution in waiting rooms: "The Problems and Myths of the Health Care System in the United States," "Maybe We Are Not Spending Enough!" and "10 Questions About Health Care Reform." OSMA members may order the pamphlets and a display placard at no charge. Order blanks appeared in the February issues of *OSMA News* and *Oklahoma Health Care Reform Update.* Or you can write directly to the OSMA, requesting the Health Update Series. Include your name, complete mailing address, quantity of each brochure you want, and whether to include the display placard.

■ **The controversy over whether optometrists** can perform YAG laser surgery has changed venues, so to speak. The Oklahoma Board of Medical Licensure and Supervision has sued the Oklahoma Board of Optometry Examiners, and apparently the issue now will be decided in a court of law.

■ **The Oklahoma Rural Health Association** (OKRHA) met January 27 in Oklahoma City and reelected Dr. Jay A. Gregory as its president. Dr. Gregory is currently the OSMA's president-elect. Claudia Kamas, OSMA associate director, serves on the Board of Directors. OKRHA also decided that during the current legislative session, it will make a statewide effort to educate consumers and health care providers about those bills affecting rural health care. Members plan to distribute press releases to their local newspapers and talk to civic groups about OKRHA.

■ **"Medicare: What It Will *and Will Not* Pay For," a** popular patient brochure produced by the American Society of Internal Medicine (ASIM) has been update for 1994. The brochure explains Medicare benefits and exclusions to older Americans in plain English, with easy-to-understand explanations of terms such as *limiting charges* and *recognized payment amounts.* It also provides a detailed summary of what is and what *is not* covered under hospital (Part A) and medical (Part B) insurance. Copies are available only in bulk quantities at $39.25 per 100, $179.50 per 500, or $318.25 per 1,000 (ASIM members receive a 20% discount). Send check payable to ASIM Literature Order Dept. NR, 2011 Pennsylvania Ave., NW, Suite 800, Washington, D.C., 20006, or call (202) 835-2746, ext. 253 for Visa or Mastercard charge.

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CLARITIN®
brand of loratadine
TABLETS
Long-Acting Antihistamine

BRIEF SUMMARY
(For full Prescribing Information, see package insert.)

INDICATIONS AND USAGE
CLARITIN Tablets are indicated for the relief of nasal and non-nasal symptoms of seasonal allergic rhinitis

CONTRAINDICATIONS
CLARITIN Tablets are contraindicated in patients who are hypersensitive to this medication or to any of its ingredients

PRECAUTIONS
General: Patients with liver impairment should be given a lower initial dose (10 mg every other day) because they have reduced clearance of CLARITIN Tablets

Drug Interactions The coadministration of a single 20 mg dose of CLARITIN Tablets (double the recommended daily dose) and a 200 mg dose of ketoconazole twice daily to 12 subjects resulted in increased plasma concentrations of loratadine (180% increase in AUC) and its active metabolite, descarboethoxyloratadine (56% increase in AUC). However, no related changes were noted in the OTc on ECGs taken at 2, 6, and 24 hours after the coadministration of loratadine and ketoconazole. Also, there were no significant differences in clinical adverse events between CLARITIN Tablet groups with or without ketoconazole

Other drugs known to inhibit hepatic metabolism should be coadministered with caution until definitive interaction studies can be completed. The number of subjects who concomitantly received macrolide antibiotics, cimetidine, ranitidine, or theophylline along with CLARITIN Tablets in controlled clinical trials is too small to rule out possible drug-drug interactions. There does not appear to be an increase in adverse events in subjects who received oral contraceptives and CLARITIN Tablets compared to placebo

Carcinogenesis, Mutagenesis, and Impairment of Fertility In an 18-month oncogenicity study in mice and a 2-year study in rats, loratadine was administered in the diet at doses up to 40 mg/kg (mice) and 25 mg/kg (rats). In the carcinogenicity studies pharmacokinetic assessments were carried out to determine animal exposure to the drug. AUC data demonstrated that the exposure of mice given 40 mg/kg of loratadine was 3.6 (loratadine) and 18 (active metabolite) times higher than a human given 10 mg/day. Exposure of rats given 25 mg/kg of loratadine was 28 (loratadine) and 67 (active metabolite) times higher than a human given 10 mg/day. Male mice given 40 mg/kg had a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) than concurrent controls. In rats, a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) was observed in males given 10 mg/kg and males and females given 25 mg/kg. The clinical significance of these findings during long-term use of CLARITIN Tablets is not known

In mutagenicity studies, there was no evidence of mutagenic potential in reverse (AMES) or forward point mutation (CHO-HGPRT) assays, or in the assay for DNA damage (Rat Primary Hepatocyte Unscheduled DNA Assay) or in two assays for chromosomal aberrations (Human Peripheral Blood Lymphocyte Clastogenesis Assay and the Mouse Bone Marrow Erythrocyte Micronucleus Assay). In the Mouse Lymphoma Assay, a positive finding occurred in the nonactivated but not the activated phase of the study

Loratadine administration produced hepatic microsomal enzyme induction in the mouse at 40 mg/kg and rat at 25 mg/kg, but not at lower doses

Decreased fertility in male rats, shown by lower female conception rates, occurred at approximately 64 mg/kg and was reversible with cessation of dosing. Loratadine had no effect on male or female fertility or reproduction in the rat at doses of approximately 24 mg/kg

Pregnancy Category B There was no evidence of animal teratogenicity in studies performed in rats and rabbits. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, CLARITIN Tablets should be used during pregnancy only if clearly needed

Nursing Mothers Loratadine and its metabolite, descarboethoxyloratadine, pass easily into breast milk and achieve concentrations that are equivalent to plasma levels with an $AUC_{0-\infty}$, AUC_{plasma} ratio of 1.17 and 0.85 for the parent and active metabolite respectively. Following a single oral dose of 40 mg, a small amount of loratadine and metabolite was excreted into the breast milk (approximately 0.03% of 40 mg over 48 hours). A decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when CLARITIN Tablets are administered to a nursing woman

Pediatric Use Safety and effectiveness in children below the age of 12 years have not been established

ADVERSE REACTIONS
Approximately 90,000 patients received CLARITIN Tablets 10 mg once daily in controlled and uncontrolled studies. Placebo-controlled clinical trials at the recommended dose of 10 mg once a day varied from 2 weeks to 6 months duration. The rate of premature withdrawal from these trials was approximately 2% in both the treated and placebo groups

REPORTED ADVERSE EVENTS WITH AN INCIDENCE OF MORE THAN 2% IN PLACEBO-CONTROLLED ALLERGIC RHINITIS CLINICAL TRIALS
PERCENT OF PATIENTS REPORTING

	LORATADINE 10 mg QD n = 1926	PLACEBO n = 2545	CLEMASTINE 1 mg BID n = 536	TERFENADINE 60 mg BID n = 684
Headache	12	11	8	8
Somnolence	8	6	22	9
Fatigue	4	3	10	2
Dry Mouth	3	2	4	3

Adverse event rates did not appear to differ significantly based on age, sex, or race, although the number of non-white subjects was relatively small

In addition to those adverse events reported above, the following adverse events have been reported in 2% or fewer patients

Autonomic Nervous System Altered salivation, increased sweating, altered lacrimation, hypoesthesia, impotence, thirst, flushing

Body As A Whole Conjunctivitis, blurred vision, earache, eye pain, tinnitus, asthenia, weight gain, back pain, leg cramps, malaise, chest pain, rigors, fever, aggravated allergy, upper respiratory infection, angioneurotic edema

Cardiovascular System Hypotension, hypertension, palpitations, syncope, tachycardia

Central and Peripheral Nervous System Hyperkinesia, blepharospasm, paresthesia, dizziness, migraine, tremor, vertigo, dysphonia

Gastrointestinal System Abdominal distress, nausea, vomiting, flatulence, gastritis, constipation, diarrhea, altered taste, increased appetite, anorexia, dyspepsia, stomatitis, toothache

Musculoskeletal System Arthralgia, myalgia

Psychiatric Anxiety, depression, agitation, insomnia, paroniria, amnesia, impaired concentration, confusion, decreased libido, nervousness

Reproductive System Breast pain, menorrhagia, dysmenorrhea, vaginitis

Respiratory System Nasal dryness, epistaxis, pharyngitis, dyspnea, nasal congestion, coughing, rhinitis, hemoptysis, sinusitis, sneezing, bronchospasm, bronchitis, laryngitis

Skin and Appendages Dermatitis, dry hair, dry skin, urticaria, rash, pruritus, photosensitivity reaction, purpura

Urinary System Urinary discoloration, altered micturition

In addition, the following spontaneous adverse events have been reported rarely during the marketing of loratadine: peripheral edema, abnormal hepatic function, including jaundice, hepatitis and hepatic necrosis, alopecia, seizures, breast enlargement, erythema multiforme, and anaphylaxis

OVERDOSAGE
Somnolence, tachycardia, and headache have been reported with overdoses greater than 10 mg (40 to 180 mg). In the event of overdosage, general symptomatic and supportive measures should be instituted promptly and maintained for as long as necessary

Treatment of overdosage would reasonably consist of emesis (ipecac syrup), except in patients with impaired consciousness, followed by the administration of activated charcoal to absorb any remaining drug. If vomiting is unsuccessful, or contraindicated, gastric lavage should be performed with normal saline. Saline cathartics may also be of value for rapid dilution of bowel contents. Loratadine is not eliminated by hemodialysis. It is not known if loratadine is eliminated by peritoneal dialysis

Oral LD_{50} values for loratadine were greater than 5000 mg/kg in rats and mice. Doses as high as 10 times the recommended clinical doses showed no effects in rats, mice, and monkeys

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Reference
1. Bedard P-M, Del Carpio J, Drouin MA, et al. Onset of action of loratadine and placebo and other efficacy variables in patients with seasonal allergic rhinitis. *Clin Ther. 1992;14:268-275.*

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YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

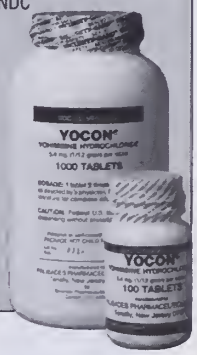
Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.



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3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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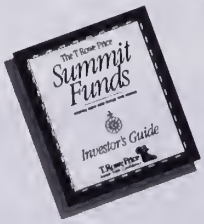

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Guest Editorial

A Plea

Dr. Nicklas' article "Whores Are Us" [page 185] points out, in graphic and understandable clarity, a facet of medical practice distasteful to many of us. With increasing frequency we see our colleagues descend to levels of conduct which would have, not too many years ago, led to censure and contempt. We see too many nonphysicians wanting (and often succeeding in these efforts) to practice medicine. Even now the optometrists are conducting a concerted effort to gain legality for laser (surgical!) procedures and expanded prescribing practices. Advertising and practice promotion activities have begun to reduce our public image from that of a noble profession to that of trades people and hucksters. Continued without restraint these activities can do irreparable harm to medicine. Please, please, PLEASE! Let us not allow ourselves to descend to these depths of commercialism. Let's be neither whores nor johns.

—Ollie W. Dehart, MD
Vinita

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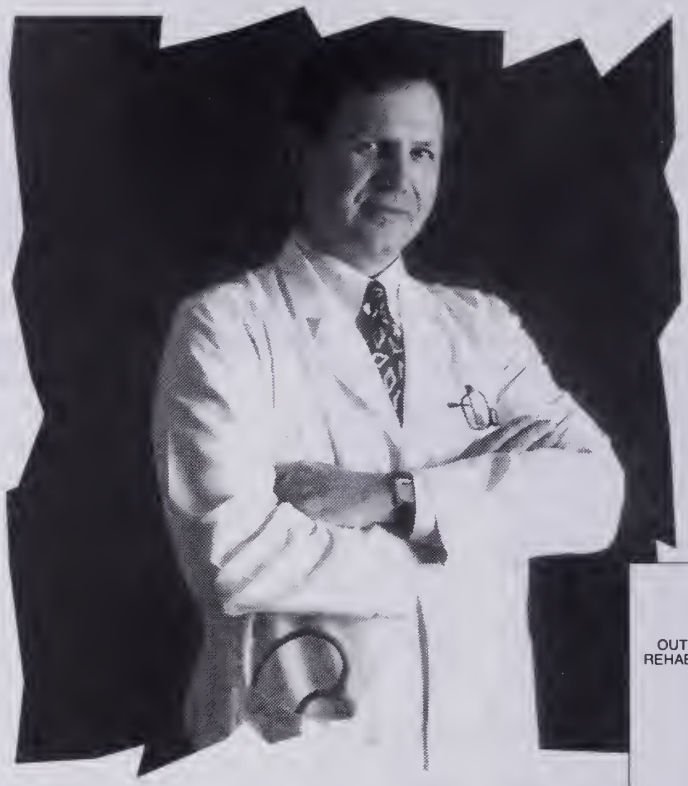

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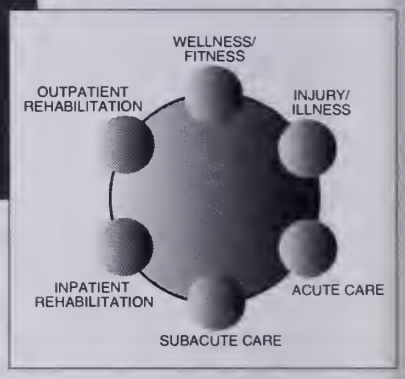

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President's Page

Jay A. Gregory, MD

"Are We There Yet, Daddy?"

How many times, as parents, have we heard that question? During the two-hour drive home from meetings in Oklahoma City over the past several years, I have often asked myself that question. So I will officially ask you, Dr. Miller, "Are we there yet, Daddy?"

I know the painful answer. But I was so hoping that the work on the managed care organization and the IPA would be finished, so I would have just the worries of the day-to-day activities of the association; the worries of national and state health system reform; the worries of specialists versus primary care; the worries of alliances invading our practices and robbing us of our market share (i.e., patients); the splintering of our efforts by those self-centered or self-interest groups; the worries over the loss of the "all for one and one for all" attitude; the worries of how to make this organization more responsive to the needs of the membership; the worries about the tremendous lack of rank and file input and participation in all facets of the association; the worries about the education of our children, the health of our children, and the violence to which our children and all Oklahomans are exposed; AIDS; cancer; heart disease; the job of keeping the AMA responsive to your needs and the needs of our patients; and the job of keeping such groups as the American College of Surgeons in harmony with the needs of all physicians... to name but a few.

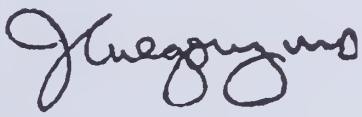
Yes, unfortunately, my friends, I know the answer to the question.

Do not think for a moment that my posing the question in such a way implies that any one of us is a parent and the rest of us children. We all are children. We all are equal. We all have something to contribute to the betterment of the whole. The challenge I have for you is, are you ready to bring to the table your contribution in a constructive, considerate manner?

In taking on the responsibility of the office of president of your OSMA, I am humbled to follow in the footsteps of so may of the great physicians and thinkers in Oklahoma medicine. I will always cherish those conversations with Dr. Miller and all the other past presidents during my formative years. I certainly look forward to the input I know is to come from all of you in the months ahead. My pledge to you is that I will always listen and consider your input before any decision is made. But it is going to be your responsibility to assure that the leadership gets your input in a timely and constructive fashion.

My pledge to you is that I will always listen and consider your input before any decision is made.

I pray that come April 1995, when we ask ourselves, "Are we there yet, Daddy?" each of us will be able to look back with pride to that small but significant part we played in getting a little closer to our destination. So let's continue the work that is before us, and make this state a better place for our children, our families, our patients, and our fellow physicians.



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Perinatal Mortality in Oklahoma

Warren M. Crosby, MD

Objective: To identify areas of need in Oklahoma's perinatal care system by analysis of trends in perinatal mortality from 1939 through 1991. *Data:* Derived from birth certificates and annual tabulation published by the Maternal Child Health Division, Oklahoma State Department of Health. *Results:* Every measure of perinatal mortality has declined in the period studied. *Conclusions:* (1) Oklahoma's perinatal mortality rate is declining. (2) Preventable deaths still occur. (3) Programs are in place to appropriately deal with the problems that lead to preventable deaths. (4) These programs must be allowed to continue or expand their efforts to further reduce preventable perinatal death.

PERIODIC REVIEW OF PERINATAL AND INFANT MORtality allows state and medical officials to evaluate the results of programs whose goals include improvement in the outlook for successful completion of pregnancy and for children during their first year of life. This study reviews the trends in each of the statistical components of perinatal and infant mortality and the possible influence of some programs on these components, and makes suggestions as to how Oklahoma may continue to improve the outlook for its children.

Perinatal care in this article is used in its broadest sense, encompassing care from the initiation of pregnancy through the baby's first year of life.

Direct correspondence to Warren M. Crosby, MD, Department of Obstetrics and Gynecology, University of Oklahoma Health Sciences Center, Post Office Box 26901, Oklahoma City, OK 73190.

Methods

The data graphs and tables were derived from "Selected Demographic Information" published annually by the Maternal and Child Health Service of the Oklahoma State Department of Health (OSDH). The data are derived from birth certificates. Data regarding the ranking of the various states were calculated from infant mortality statistics published by the National Center for Health Statistics.

Definitions

Live birth: the birth of a child that shows any evidence of life after it is born. (Note: this may include fetuses born before 20 weeks gestation.)

Fetal death: a fetus born after the beginning of the 20th week of gestation and not alive at birth.

Neonatal death: death of a child born alive that dies within the first 28 days following birth.

Perinatal deaths: the sum of fetal deaths and neonatal deaths.

Post neonatal death: death of a child between its 28th day of life and its first birthday.

Infant death: death of a child within its first year (equal to the sum of neonatal deaths and post neonatal deaths).

Each of these defined terms may be expressed as a *rate,* in which the number of deaths are divided by the number of live births. Thus, the infant mortality rate is defined as the number of infant deaths occurring in a given year divided by the number of live births over the same period of time, expressed as the number of deaths per 1,000 live births. Calculation

of rates allows comparisons of mortality risks between various entities, such as states or countries. Each rate may be said to relate to specific problems[1]:

The fetal mortality rate is generally thought to relate to the quality of medical care for high risk problems that complicate pregnancies in which medical care of the mother may ameliorate the effects of the maternal disease on the fetus. In diseases such as maternal diabetes mellitus or Rh immunization, the quality of perinatal care correlates well with the fetal death rate.

The neonatal mortality rate is most dependent upon the length of gestation and the birth weight of the newborn. Thus, "weight-specific" death rates are useful when comparing neonatal death rates from other populations. The dramatic decline in neonatal death rates in the United States in recent years reflects the improvement in the care of the small newborn and, in particular, the management of the respiratory distress syndrome.

The perinatal mortality rate is thought to reflect a combination of the health of the obstetric population and the quality of obstetric and newborn care.

The post neonatal mortality rate is thought to reflect the influences of the infant's home environment more than influences originating during pregnancy. Post neonatal death is most often caused by congenital anomalies, infectious disease, SIDS, trauma and, somewhat indirectly, premature birth. Post neonatal mortality is now nearly equal to the neonatal rate in Oklahoma.

The infant mortality rate (IMR) is the rate most frequently used to compare one population or geographic area with another with regards to the overall quality of perinatal care and general health of mothers in the reproductive age groups. (Congenital anomalies, a common cause of death in each category, are not considered preventable for the purposes of this communication.)

Innumerable factors, known and unknown, may contribute to the death of the fetus or infant. The perinatal or infant mortality rate derived from the actual number of deaths represents not only the sum of all of these factors, but random variation as well. Thus, while we use such rates to draw certain conclusions about the populations studied and the influence of medical care on the population, it is clear that such conclusions may be inaccurate or misleading. Keeping this is mind, it is still useful to calculate mortality rates, if for no other reason than to identify areas or populations in apparent need of attention, and to direct that attention to those problems whose solution is most likely to improve preventable mortality.

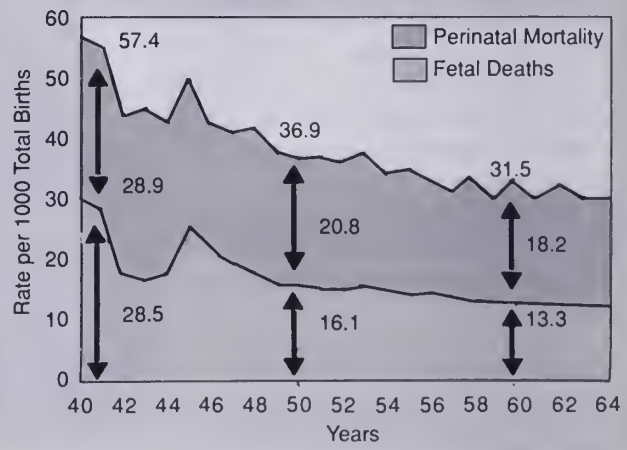
Data Analysis

In 1965 the OSDH published a monograph prepared by John Shackelford, MD, and Mary I. Ward, statistician, entitled "Perinatal Mortality Trends in Oklahoma."[2] This publication covered Oklahoma birth certificate data from 1939 through 1965 and provides a background for this review. Figure 1 depicts the perinatal mortality for that time period, divided into fetal deaths and neonatal deaths, which total perinatal deaths. Dr. Mosley, commissioner of health and the author of this monograph, praised the trend toward delivering in hospitals, noting that it was not until 1943 that more than half of Oklahoma births took place in a hospital; by 1964 over 98% did so. Dr. Mosley hoped that the monograph would arouse public awareness of the need for "a continuum of adequate medical supervision of mothers before, during, after, and between pregnancies" and that it would promote "projects to extend diagnostic services and routine care for all mothers and newborn infants and intensive care for high risk groups."

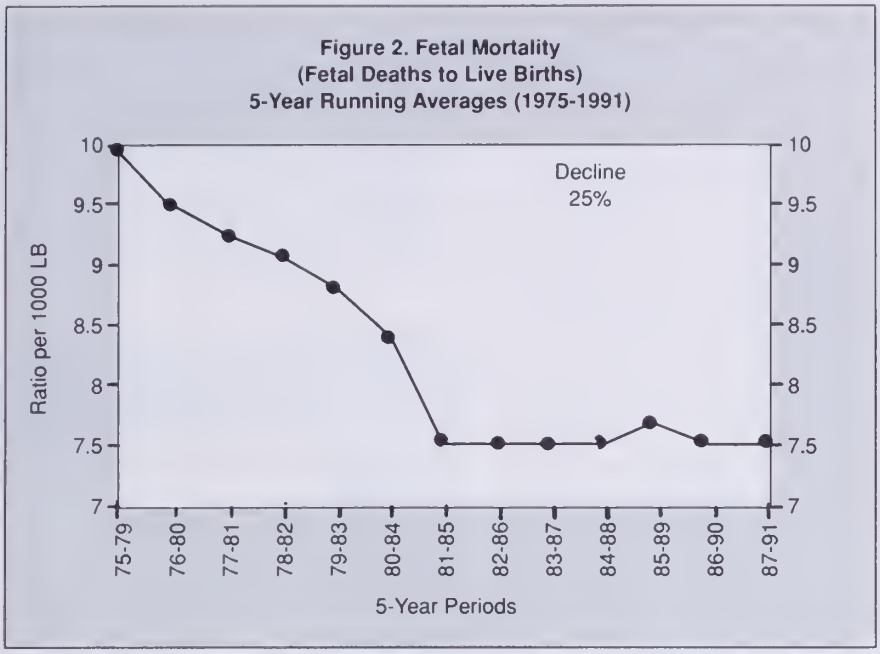
The data graphed in Figures 2 through 9 were derived from birth certificates, but reported as five-year running averages. This technique smooths out wide variations in data from year to year so the trends in the raw data are made more apparent.

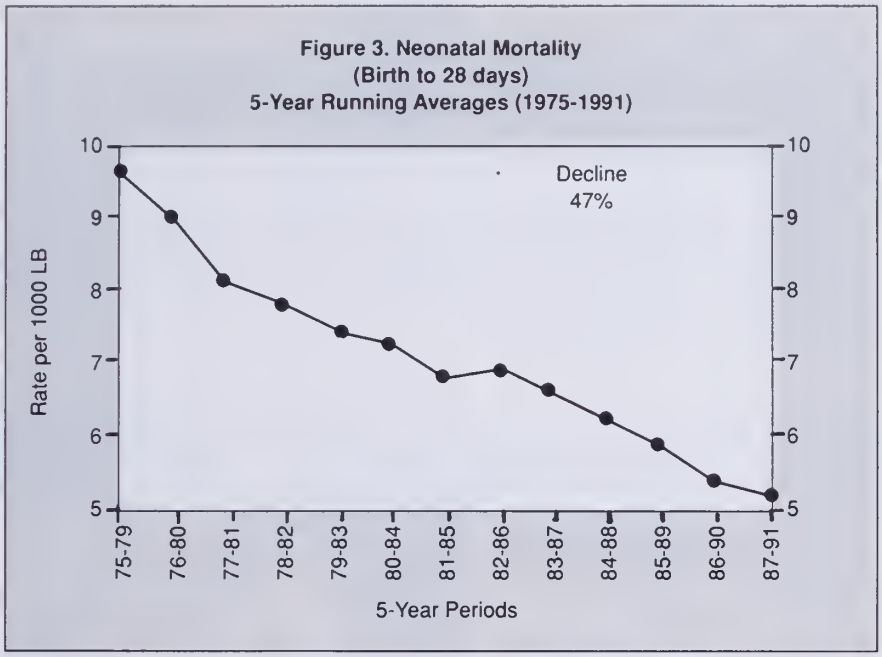
Data on fetal mortality (Fig. 2) is included partly because an improvement in the rate reflects an improvement in perinatal care. It may also reflect the rate of very high risk pregnancies and congenitally

Figure 1. Reduction in Perinatal Mortality Rates Oklahoma 1939-1964



abnormal fetuses for which improved medical care would have no benefit. When considering overall infant mortality, the fetal death rate is an important consideration because a low infant mortality rate in a group of high risk pregnancies (i.e., diabetes, hypertension. Rh isoimmunization) might actually be a reflection of poor prenatal care in which the maternal disease was not treated and fetal death resulted in many pregnancies before the onset of labor. Under such circumstances, the infant mortality rate, which includes only live born infants, would be lower than a similar population of high risk women who received better prenatal care and delivered living babies who later succumbed unavoidably to the influence of the mother's disease. Knowledge of the fetal death rate is important when drawing conclusions as to quality of care from the infant mortality rate. In Oklahoma, fetal mortality declined 21% between 1975 and 1991.





The neonatal mortality rate (Fig. 3) bears a direct relationship to the incidence of congenital anomalies and the birth weight of individual babies. The 47% improvement from 1975 through 1991 (Fig. 3) is most likely due to the dramatic improvement in the care of small and/or sick newborns. There has been a decline in the incidence of preterm birth from seven to six percent over this time period (Table 1), but this is not nearly enough to account for the more substantial decline in overall neonatal mortality.

With a given level of medical care for all pregnant women and their newborns, the perinatal mortality rate (Fig. 4) may reflect the general quality of health in the community which, in turn, is influenced by socioeconomic and racial factors. For instance, the black population of the United States has a significantly higher infant and perinatal mortality rate than does the white.[1] There are some indications that this is not entirely due to poor socioeconomic or generally poor health status in the black population. Conversely, given a population stable with respect to demographic characteristics and levels of disease, perinatal mortality may relate directly to the quality of perinatal care. Oklahoma's perinatal mortality rate declined by 33% from 1975 through 1991,

(continued)

reflecting improvement in both fetal death and neonatal death rates during this period.

Post neonatal mortality is thought to reflect the influence of environmental factors such as infectious disease and trauma. The availability and quality of medical care is related to timely and appropriate treatment for serious infectious disease. This statistic is also useful as an indication as to whether neonatal care simply prolongs life beyond the 28th day. If that were to happen to a significant number of newborns, the post neonatal mortality rate would rise while the neonatal mortality rate was declining. Oklahoma's post neonatal mortality rate since 1975 is depicted in Figure 5.

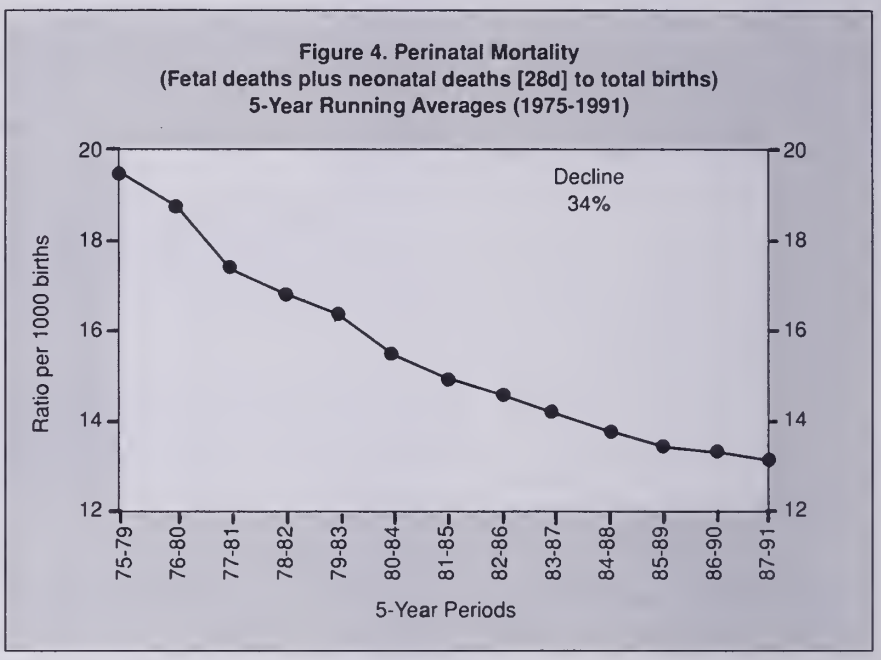
While the decline in post neonatal mortality has been 20% since 1975, the number of post neonatal deaths represents about half of all infant deaths, compared to about one-third in most states.

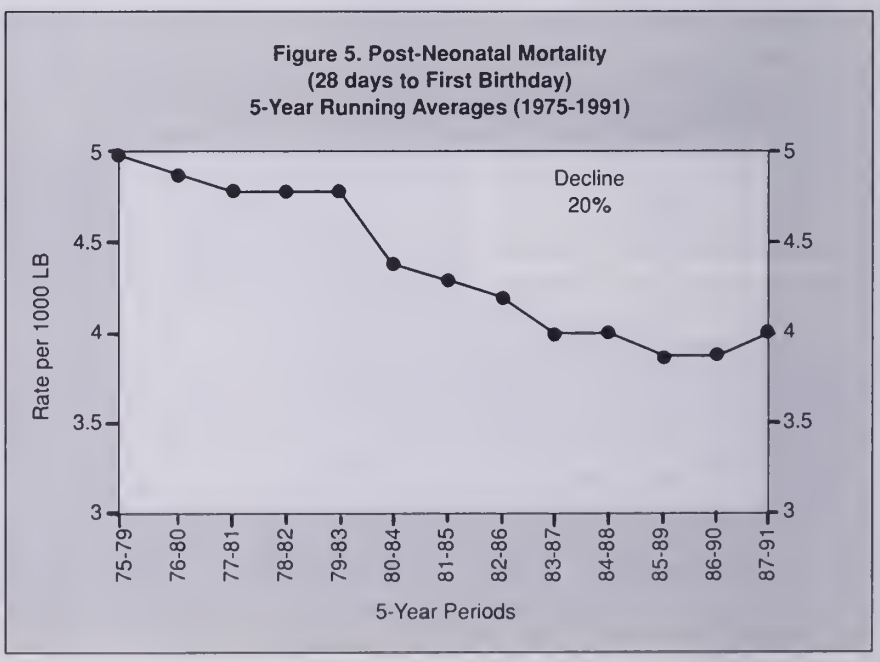
The infant mortality rate (Fig. 6) reflects the general health of babies and the care they receive when they are born sick or acquire medical problems after birth (but within the first year of life). Great strides have been made in prenatal and newborn care and thus the IMR has continued to decline. Figure 6 shows an improvement from 14.5 to 9.2 infant deaths per 1,000 live births from 1975 to 1991, a 30% decline.

Comparative Rank Among the States.— Rank among states (Table 2) is one way to compare one state's programs with another. The comparison is relatively crude, however, because each state is unique with regards to the myriad factors that contribute to infant mortality. Furthermore, the rates tend to be very close together, and for many states, a dif-

Table 1. Adequacy of Care and Low Birth Weight

Epoch	% Adequate Prenatal Care	% Late Little or No Care	% First Trimester Care	% Low Birth Wt
1972-76	47	11	N/R	7.6
1977-81	50	11	69	7.0
1982-86	53	10	69	6.5
1987-91	58	8	73	6.6





ference of fewer than 10 infant deaths may change the rate by 0.1 per 1,000 live births. With 50,000 live births per year in Oklahoma, 15 or 20 more infant deaths in a given year can change the states' ranking considerably. For instance, in 1989, Oklahoma had 394 infant deaths from 47,238 live births, an IMR of 8.3. In that year, there were 13 states with a lower IMR than Oklahoma. Oklahoma thus ranked 14th in the United States. However, a year later, the fifth year chosen for the five-year comparisons depicted in Table 2, Oklahoma had 489 more live births (47,725) and 38 more infant deaths (432). The resultant IMR in 1990 was 9.1, which placed Oklahoma 28th among the states.

Largest Metropolitan Counties.— Figure 7 shows the year-by-year infant mortality rates for Tulsa, Oklahoma, and Comanche counties from 1975 through 1991. The rates vary considerably from year to year, but there has been a definite downward trend for all three, particularly since 1984. The average rate for each county and in the aggregate exceeds the rate for the entire state throughout the time period.

It is probable that the IMR in these counties has responded to a number of formal and informal community efforts to enhance availability, access, and quality of perinatal care developed in these counties in the early and middle 1980s.

In a recently published analysis of the IMR data for U.S. cities of over 250,000 population that covered the years 1984–1989, Oklahoma City is second only to Washington, D.C., in the IMR of its white population. The IMR for the Oklahoma City black population ranks

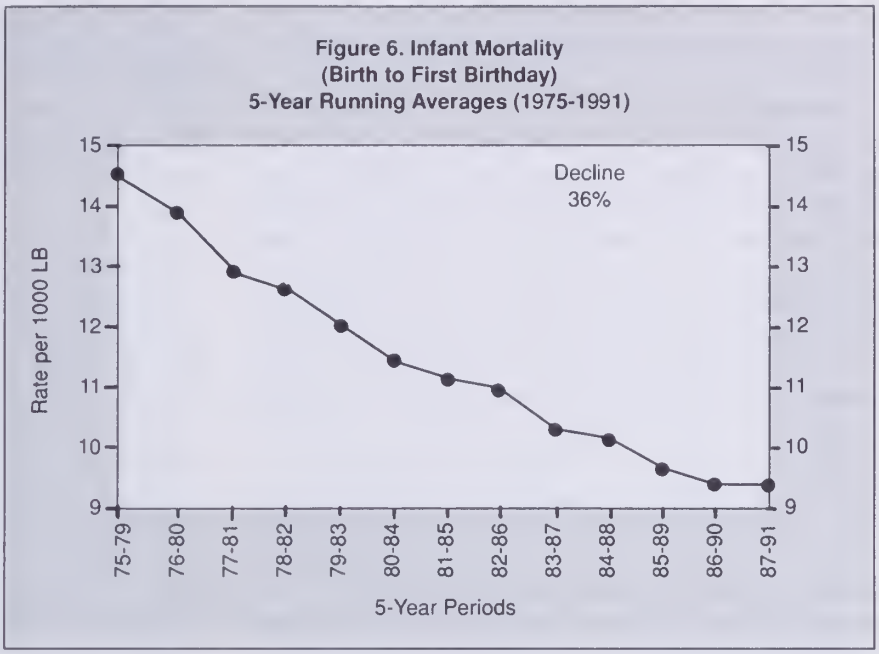
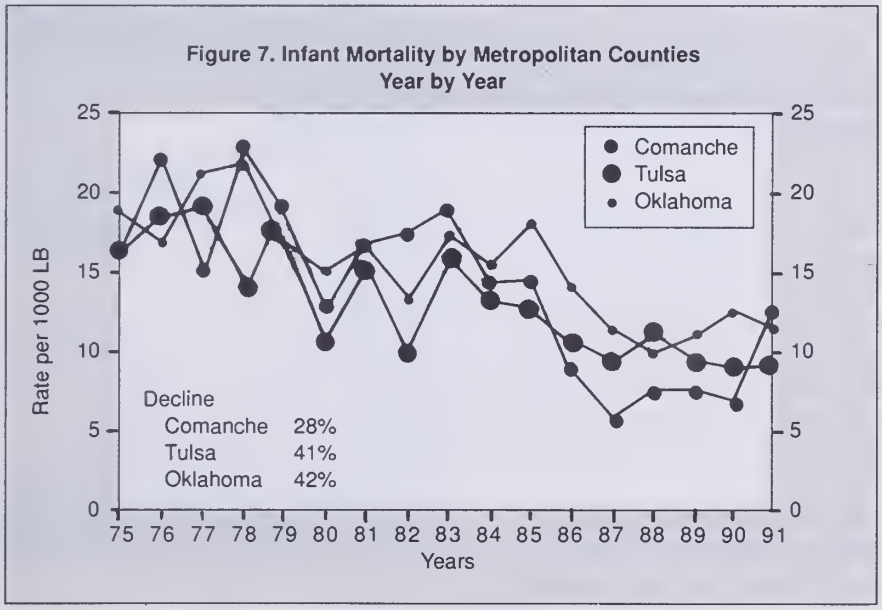


Table 2. Oklahoma Ranking According to IMR

Year	OK IMR	Rank	WSC*	US	Best	Worst
1970	21.3	33	22.2	20.0	14.3 (ND)	28.5 (MS)
1975	16.2	29	17.3	16.1	12.9 (HI)	22.3 (MS)
1980	12.7	33	13.0	12.6	9.2 (ME)	17.0 (MS)
1985	10.9	30	11.1	10.6	8.2 (RI)	13.7 (MS)
1990	9.1	28	8.6	9.2	6.4 (VT)	12.2 (MS)

*West South Central Region (AR, LA, OK, TX)



33rd amongst the larger cities, which nearly equals Oklahoma City's rank in size.[3]

Comment

The good news is that Oklahoma is improving the outlook for successful completion of its citizens' pregnancies. The downward trend in infant, perinatal, fetal, neonatal, and post neonatal mortality is clear and important. The improvement reflects a variety of influences, only some of which can be identified. The general level of health of reproductive age women in Oklahoma may be improving, but there are no data to support or refute the concept. Increasing use of contraceptive and abortion services certainly reduces the number of unwanted pregnancies. Unwanted infants have been shown to have higher mortality rates than wanted ones.[4] Poverty also is associated with a group of factors related to high infant mortality; poor nutrition, poor housing, substance abuse, and less medical and prenatal care. On a national scale, Oklahoma is among the 10 states with the highest number of children living in poverty.[5]

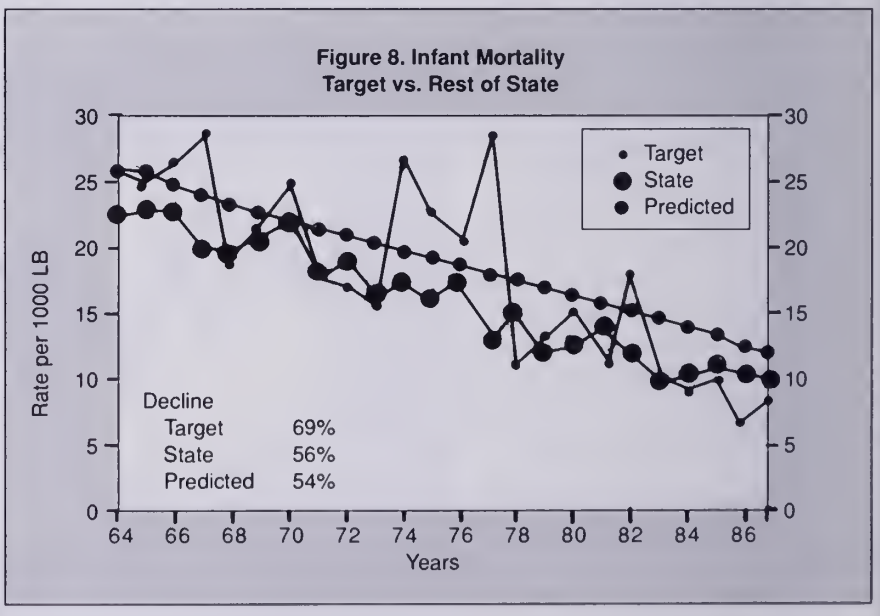
The state is doing as much as it is willing to do to improve the economic status of its poorest citizens. Thus, the likelihood of eliminating poverty as a factor contributing to infant mortality is remote. However, several programs apparently have had a salutary effect on infant mortality in Oklahoma. The Improved Pregnancy Outcome (IPO) program was developed by the Bureau of Maternal and Child Health of the U.S. Department of Health and Human Services, and is managed in Oklahoma by OSDH. Among other things, this program increased access to prenatal care in many counties by instituting prenatal care in county health clinics where it had not been available before. The Oklahoma Rural Infant Care Project (ORICP), a program initiated by the Robert Wood Johnson Foundation, began in 1980. ORICP was a cooperative effort of the University of Oklahoma's colleges of medicine in Oklahoma City and Tulsa, and OSDH. This project involved six rural counties with high infant mortality that were already involved in IPO.

The combination of projects was associated with an impressive decline in infant mortality in the six target counties when compared with the rest of the state (Fig. 8). The starred line shows the computer-derived predicted rate of decline in IMR for the target counties based on their trend in IMR prior to 1980. The actual rate for the target counties (small squares) is considerably lower than the predicted rate. This implies that the program contributed to the lower rate.

Prenatal care programs developed locally in Tulsa, Oklahoma, Comanche, and other counties to increase access to prenatal care were developed with and continue to utilize support from OSDH. The largest of these are in Tulsa and Oklahoma City, where the departments of obstetrics and gynecology at the Tulsa Medical College, OSU College of Osteopathic Medicine, and OU College of Medicine have collaborated with local clinics and OSDH to develop a comprehensive perinatal care system offering prenatal care, high risk pregnancy and delivery care, and contraceptive counseling.

Several other statewide programs probably have contributed to the improvement in infant mortality:

1. The Perinatal Continuing Education Program (PCEP) offers outreach education to the physician and nursing staff (delivery and nursery) of hospitals throughout the state. It is supported by state funds. Over 90 hospitals have participated, many two or more times. Over 600 physicians and 3,000 nurses have completed the program. PCEP has been demonstrated to improve hospital care practices in the care of mothers and infants.[6] Northeastern Oklahoma also



has received an outreach perinatal education program (STEP) provided by St. Francis Hospital, Tulsa.

2. *Instruction in Neonatal Resuscitation*—the NRP program that teaches newborn resuscitation throughout the state—is provided by the American Academy of Pediatrics and the American Heart Association.

3. *Healthy Futures.* In 1988, a grant for the Healthy Futures Program was awarded to the Oklahoma secretary of human services by the Robert Wood Johnson Foundation. This program was developed with input from the successes and difficulties encountered by previous statewide programs (IPO and ORICP) whose funding had ceased. Its concept is to remove barriers and improve access to perinatal care services in communities throughout the state. Rural area perinatal coordinators (APCs) are responsible for 15 counties each. Tulsa and Oklahoma counties each have one APC to themselves. The APCs have prepared a needs assessment for each of their counties, outlining strengths and weaknesses with suggestions for improvement. The Healthy Futures Steering Committee is served by the APCs and the program director, with offices at the OSDH. Healthy Futures strives to identify problems in the delivery of perinatal care and to help develop solutions with the cooperation of local and state health authorities, local or regional businesses, churches, and other organizations. Solutions to problems which remain difficult are referred to the steering committee, whose membership encompasses a broad spectrum of expertise and experience. Healthy Futures is now entirely supported by state funds and has recently broadened its scope to include preventive services to newborns and older children. This program has come under recent criticism and investigation, but the APCs continue to serve their communities. The steering committee has not met since August 5, 1993.

4. *Telecommunications.* Telecommunication systems help rural communities communicate with tertiary care centers by telephone, direct telephone hook-ups with fetal monitor machines, and telefacsimile equipment.

5. *Medical Transportation.* Two medical helicopter services operating out of Tulsa (Lifeflight and AirEvac) and Oklahoma City (MediFlight), along with Oklahoma's Emergency Medical Services (EMS) ambulances, have increased timeliness and effectiveness of perinatal transports of both mothers and newborns.

These contributing programs and services form a voluntary system of perinatal care in Oklahoma that has made a major contribution to improvement in perinatal mortality in the past 15 years.

The bad news is that significant problems remain. Oklahoma's decline in infant mortality has been dramatic, but our rank among the states, even those in our immediate area, has changed little over time. This information indicates that Oklahoma continues to have preventable infant mortality and room for improvement.

Oklahoma's rate of adequacy of prenatal care (Table 1) has improved from 47% to 58% from 1972 to 1991. Since over 70% of new mothers now report beginning care in the first trimester (thus enabling them to meet the criteria for adequate care if they continue to follow recommended frequency of prenatal visits), it is clear that 10% to 15% of pregnant women drop out of care for a while during their pregnancies. The reasons for this phenomenon are not clear. It is known that a relationship exists between low socioeconomic status, low education levels, and minority race and inadequate levels of prenatal care. It is possible that women make a "first visit" during the first trimester in which the pregnancy is documented. In women without insurance or a welfare card, it may take several months to find care. The loss of visits during this time reduces the total number of visits and thus many women who were seen in the first trimester still may fail to meet the criteria for adequate care. There is a clear relationship between inadequate care and increased IMR.

Black infant mortality remains nearly double that of white infants, a relationship that has been present in Oklahoma since at least 1939. Figure 9 shows the dramatic difference between white and black infant mortality in Oklahoma.

Ten counties in Oklahoma have no prenatal or delivery care services within the county. Thirty-eight counties are listed as "medically underserved areas" by the U.S. Public Health Service.[7]

NEEDS ASSESSMENT.— Both the initial effort that culminated in 1984 with the OSDH publication of *Caring for Pregnant Women and their Infants: A Needs Assessment*[8] and *A State Plan*[9] and that that was published in 1992 by Healthy Futures[10] provide a clear outline of what is necessary to continue improving the outcome of pregnancy. These priorities have not changed.

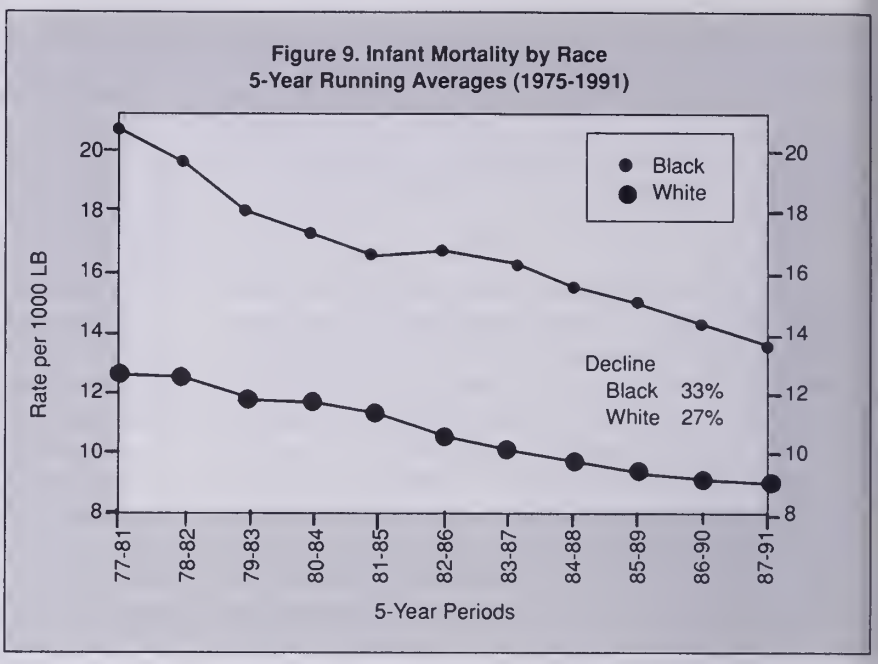
They include:

1. *Public Education.* Women of reproductive age must know that early and continuous prenatal care

will help them remain healthy and improve the health of their children.

2. Barriers to Obtaining Prenatal Care. These must be eliminated as much as possible, so that once the woman identifies herself as being pregnant, she can quickly and with minimal effort enter prenatal care and remain there.

3. Health Care Providers. Doctors and clinics must welcome all pregnant women and infants to their care. This implies reasonable if not a totally satisfactory reimbursement for their services. Neither hospitals, physicians, nurses, nor clinics can continue to provide perinatal care at a loss. Recent increases in Medicaid reimbursement to physicians may result in more women being accepted for prenatal and delivery care in hospitals near their homes.



4. Quality of Care. The quality of care given by the health care system should be high and appropriately up to date.

5. Organization. The system of perinatal care must be organized appropriately so that the funds expended are not wasted. Organization of perinatal care implies that pregnant women receive unhindered access to care near their home so long as that care is appropriate to their medical needs. Consultations, referral, and transportation must be readily available when the need arises.

6. Infant Mortality Studies. In response to the concentration of white infant deaths in Oklahoma City, the Oklahoma Fetal and Infant Mortality Analysis Program (OFIMAP) was developed. The program has enlisted the cooperation of physicians, hospitals, and other health care providers who review the prenatal and postnatal records of all pregnancies associated with an infant death. The de-identified abstracts are discussed by technical review panels and a community action panel made up of citizens and health providers interested in influencing infant mortality. Innovations in programs, better access to care, and improvements in pregnancy outcome and child health are expected from this process. Other large counties or groups of counties may want to initiate a fetal and infant mortality analysis in the future.

7. Infant Follow-Up. A system of tracking and follow-up of high risk newborns is important to reducing the death of infants after they go home from the hospital or NICU. While SIDS remains a common cause of post neonatal death, no solution for this problem has surfaced. Immunization, access to pediatric care, and follow-up of at-risk newborns can reduce preventable deaths and each must be expanded for Oklahoma to be as successful in reducing post neonatal mortality as we have been with perinatal mortality.

Recommendations

Mechanisms to address each of these continuing priorities are in place today. Healthy Futures has the capability and experience in public education not only through broadcast and print media, but also through the APCs working with local school boards and individual teachers. The APCs are also working with each community to remove barriers and enhance access to prenatal care. This effort is beginning to have an impact. Healthy Futures must be allowed to continue and expand these important activities.

Medicaid reimbursement for perinatal care increased by $250 per pregnancy in 1992. Delays in reimbursement for physicians and hospitals are increasing. The legislature must continue increased Medicaid funding for perinatal care and to acquire attendant federal matching funds. It has been proven over and over again that for each dollar spent on prenatal care, three to four dollars are returned as reduced expenditures for newborn intensive care and chronic care for handicapped children.[9-11]

The quality of perinatal care delivered throughout the state is improving. But perinatal care is constantly changing; the standards of care just a few years ago may be inadequate today. It is not possible for rural physicians and hospitals to keep up with these changes by themselves. Continued support of outreach education programs offers the most cost-effective way for physicians and hospitals to continue to evolve and improve the quality of perinatal care.

The organization of perinatal care in Oklahoma remains an informal one, but its effectiveness continues to improve. Outreach education has fostered the concept of risk assessment for pregnant women. When risk assessment is combined with easy access to consultation and referral, patients can receive their care in the setting as close to their home as is medically and geographically appropriate. The establishment of helicopter transport systems has enhanced the timeliness and quality of transports both to and from tertiary care centers for both mothers and newborns.

In 1991, the Maternal Care Act (H.B. 1708) designated the Healthy Futures Steering Committee as the official body responsible for making recommendations for improvements in perinatal care to both houses of the legislature. The committee must be allowed to continue its activities, and should be encouraged to formalize and expand the organization of perinatal care in the state so that perinatal care expenditures may be used efficiently.

The identification and follow-up of infants at high risk can be effective in reducing post-neonatal morbidity and mortality. Programs in Tulsa (The Parent-Child Center) and Oklahoma City (The Infant Transition Project) are voluntary programs that provide follow-up for high-risk newborns. These efforts could be better funded so that their coordinated efforts can be expanded statewide.

Conclusions

Oklahoma's infant mortality rate is declining. But preventable infant deaths still occur.

Programs are in place to appropriately deal with the problems that lead to preventable deaths.

These programs must be allowed to continue their efforts, and several should be expanded so they can benefit all Oklahomans regardless of where they live.

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The Author

Warren M Crosby, MD, is a professor of obstetrics and gynecology at the University of Oklahoma Health Sciences Center in Oklahoma City.

Pretreatment of Viral Hepatitis Before Liver Transplantation

David H. Van Thiel, MD; Stefano Fagiuoli, MD; Paolo Caraceni, MD; Harlan I. Wright, MD; Judith S. Gavaler, PhD

The authors discuss viral hepatitis as an indication for liver transplantation and the problems encountered in trying to treat the disease while achieving a successful transplantation.

VIRAL HEPATITIS IS A MAJOR INDICATION FOR LIVER transplantation (LTx).[1,2] LTx is indicated in cases of viral hepatitis in three different clinical situations. These are in the order of the frequency with which they arise: (1) end stage post necrotic cirrhosis with or without a complicating hepatocellular carcinoma; (2) subfulminant or late onset hepatic failure; and (3) fulminant hepatic failure. The specific forms of viral hepatitis that occur in patients with each of these clinical syndromes are shown in Table 1.

The major types of viral hepatitis necessitating LTx as a consequence of post-necrotic cirrhosis are hepatitis B virus disease occurring either alone or in association with a delta hepatitis superinfection and hepatitis C virus disease. Because a common route of infection for each of these diseases is the parenteral use of drugs, these viral diseases occur not only alone but frequently co-exist in a single individual who manifests a high risk behavior pattern associated with the acquisition of viral liver disease. In these cases, the viral infection is typically chronic, is well established, often in several organs, and the viral load is considerable.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112.

In cases of fulminant hepatic failure, all of the currently recognized forms of viral hepatitis are represented. In most, the viral load of the patient is small as a consequence of either the intrinsic hepatotoxicity of the virus or a marked activation of the individual's immune system that characterizes the disease process. Moreover, the disease process typi-

Table 1. Indications for Liver Transplantation in Cases with Underlying Viral Hepatitis

I. End-State Post-Necrotic Cirrhosis — Most Common Indication
Hepatitis B
Hepatitis B + Delta
Hepatitis C
II. Fulminant Hepatic Failure — Unusual Indication
Hepatitis A
Hepatitis B
Hepatitis C
Hepatitis B + Delta
Hepatitis E
CMV
EBV
Herpes viruses
Adenovirus
Other less common forms of viral hepatitis
III. Subfulminant hepatitis and late onset hepatic failure — rare indication
Hepatitis B exacerbation due to chemotherapy or some other form of exogenous or endogenous immunosuppression
Drug induced hepatotoxicity associated with an excerbation of underlying HBV or HCV disease
HDV super-infection of a quiescent HBV infection
HCV infection
Paromyxovirus infection in children

cally, if not always, is characterized by infection with a single virus with the singular exception of a fulminant co-infection consisting of hepatitis B and delta hepatitis.

In the case of subfulminant and/or late onset hepatic failure, the disease process by definition is subacute to near chronic in nature. Unlike the situation in fulminant hepatic failure, late onset hepatic failure is characterized by a very high viral load. Although a large number of viruses can produce hepatic disease that leads to a requirement for LTx, in clinical practice hepatitis B and hepatitis C are the two most frequently responsible viral agents. The problem with both of these viruses is that a chronic carrier state exists for each and extrahepatic infection of the bone marrow, pancreas, and other tissue sites is known to occur.[3] As a result, following LTx, due to either of these two viruses, disease recurrence is almost inevitable.[4] Worse yet, for HBV disease the disease in the allograft liver is aggressive and can complete its entire natural history from infection of the graft to end stage liver disease with cirrhosis and even a hepatoma within a 2- to 3-year period. In the case of HCV disease, recurrent infection is similarly almost universal but recurrent disease, as opposed to recurrent infection, occurs in only a third.[5,6] Moreover, when it does, the disease progresses slowly as compared to HBV disease recurrence.[5,6]

Compounding the problems of a near-universal infection rate of the liver allograft and the accelerated natural history of viral hepatitis in a liver graft are the effects of immunosuppression upon viral replication and concerns about the likelihood of inducing a rejection episode with interferon (IFN) therapy, the only agent proven to be effective in cases of viral hepatitis due to either hepatitis B or hepatitis C.[7-9] Because of the magnitude of the overall problem of both HBV and HCV diseases worldwide, these two viral diseases will continue to present a problem to LTx recipients and the physicians and surgeons at LTx centers (Table 2).

Moreover, in clinical LTx it must be remembered that not only the recipient may determine the development of post-LTx viral liver disease, but so also can the donor (Table 3). It is well known that the donor population does not reflect society at large. Instead, as a population, it is characterized by being male between the ages of 20 and 40 years, impulsive and risk taking in nature. As a result of these characteristics, the donor population has a high rate of sudden unexpected death. Moreover, donors have a high rate of serologic evidence for prior viral liver disease.

Table 2. Prevalence of HBV and HCV Disease Worldwide

HBV
- Estimated incidence world wide 300 million cases.
- 75,000 cases annually in the United States.
- 25% of these become chronic with 5% developing infection.

HCV
- Estimated 600 million cases world wide.
- 150,000 cases annually in the United States.
- 50-70% of these becoming chronic and half developing cirrhosis.

Table 3. Donor Characteristics That Predispose to Viral Liver Disease in Liver Allograft Recipients

Predominantly male	
Age: 20-40 years	
Impulsive and high risk taking behavior	
HBcAb+	45%
HCV-Ab+	1.8%
CMV+	58%

Thus, data obtained from the New England Organ Bank in 1991 demonstrate that 45% of all liver donors are HBcAb+, 1.8% are HCV-Ab+, and 58% are CMV+.

Because of viral infection of the allograft, the long-term survival of individuals transplanted for HBV disease is reduced compared to that of patients transplanted for other forms of hepatocellular diseases .[4,10] While no difference is seen between those transplanted for HBV disease and other forms of hepatocellular disease in the first 6 months following LTx, after 6 months a significant difference in survival becomes evident and continues to increase through the next 18 months.[4,10] This decline in patient survival is due, almost exclusively, to allograft infection and subsequent graft failure as a consequence of progressive viral liver disease. As noted earlier, individuals who are HBsAg+ when transplanted have a very high rate of graft viral infection, as shown in Table 4. In essentially all series, the rate is greater than half those transplanted and can be as high as 90%.[4]

The source of this allograft infection is probably a combination of the circulating HBV present in the blood of the recipient at the time of LTx and the continual release into the blood of virus from extrahepatic tissue sites that also are infected.[3,4] In those recipients, who are not HBsAg+ prior to LTx but acquire the disease following LTx, the source of the infection is either the donor organ (reactivation of a latent viral infection) in a HBcAb+ donor or one of

the blood products that the recipient has received during the LTx procedure.

Following graft infection, any of three different pathways can be taken. These are: (1) fulminant hepatitis, which is quite rare; (2) a form of subacute or late onset hepatic failure termed *fibrosing cholestasis,* which is uncommon; and (3) chronic liver disease that can progress to either cirrhosis or cirrhosis plus hepatocellular carcinoma in only 1 to 3 years.[11] This is the most common outcome.

The only recognized therapy for HBV disease is α-interferon (IFN). In HBV disease it acts both as an immunomodulating agent by stimulating the cellular immune system to enhance viral clearance and, to a lesser degree, as an antiviral agent inhibiting viral replication. Because of these properties of IFN, we used it in potential liver transplant recipients both before and immediately after LTx in an attempt to prevent allograft infection and to increase overall graft and patient survival.

The characteristics of the patients enrolled in this clinical study are shown in Table 5. A total of 28 patients without hepatocellular cancer in the excised recipient liver were studied. As a group, they received IFN for a minimum of 14 days prior to LTx, receiving on mean a total of 32.3 ± 7.4 days of IFN prior to being transplanted. They continued to receive IFN for a full 6 months post-LTx. An important aspect of this study was that the immunosuppression administered to the patients post-LTx was prescribed by a team of physicians that was independent of those prescribing and monitoring the patients' hematologic parameters and adjusting the dose of IFN.

Following completion of the study, the patients could be divided into two groups based upon their clearance of HBV-DNA while receiving IFN therapy. Those that cleared their serum of HBV-DNA prior to LTx continued to be HBV-DNA negative while receiving IFN. Moreover, they had lower HBV-DNA serum levels at the time of enrollment as compared to those who failed to clear HBV-DNA prior to LTx. Both groups had either low or undetectable levels of HBV-DNA at 2 weeks post-LTx. The responders continued to be HBV-DNA free while on IFN. In contrast, those who failed to respond developed viral titers with increasing time post-LTx. The SGPT (ALT) level of the responders was lower than that of the non-responders at entry. In response to IFN therapy, it rose to a greater level than that experienced by the non-responders. Following LTx, both groups had normal SGPT levels from 2 through 6 weeks post LTx while receiving IFN.

Of the 28 patients enrolled in the study, 4 died prior to LTx or during the LTx procedure (Table 6). One of these deaths, the one due to spontaneous bacterial peritonitis, appeared to be a direct consequence of receiving the IFN therapy. One of the deaths following LTx occurred as an indirect consequence of having a liver biopsy performed as part of the study.

Table 4. Reported Rate of Allograft Infection Following Liver Transplantation of HBsAg+ Recipients

Author	Rate	Therapy
O'Grady	90%	None on HB1G
Todo	82%	Variable
Samuel	59%	HB1G (long term)
Mueller	57%	HB1G (6-12 months)
Hopf	71%	None on ST-HB1G + IFN

Table 5. Characteristics of the Patients Studied

1. All had HBV positive end-stage liver disease and were Child's class C
2. All were HBV-DNA positive prior to LTx
3. Prospectively enrolled from 11/86 to 2/89
4. All were treated with α Interferon daily at a dose ranging from 1.25 to 5.0 million units daily depending upon the patient's hematologic parameters (WBC and platelet counts) prior to LTx and for 6 weeks following LTx.

Despite these major complications of LTx and the IFN therapy, the 2-year follow-up of these patients showed a remarkable post-LTx survival of 75%. This figure compares favorably with that of control individuals transplanted for hepatocellular disease not related to HBV infection (76%) and was considerably better than that of historical controls transplanted for HBV disease who did not receive IFN therapy having a 49% survival rate ($p \leq 0.02$). The incidence of acute cellular rejection in LTx recipients treated with IFN was similar to that discussed in historical controls.

The major side effects of the IFN therapy in this group included leucopenia, thrombocytopenia, fever, headache, nausea, anorexia, delirium, and depression. The latter two necessitated a transient discontinuation of therapy and a dose reduction thereafter.

An unfortunate follow-up note to this study is the fact that although the responders were HBV-DNA negative for the 6 weeks following LTx while they were receiving IFN therapy, they all became HBV-DNA positive following discontinuation of the IFN therapy. Despite this undesirable evidence for reinfection following IFN discontinuation at 6 weeks post LTx, the benefit of IFN treatment in terms of graft and patient

Table 6. Mortality Experienced During the Course of Therapy in HBV LTx Recipients

1. Pretransplant deaths ($n = 3$)
   Variceal bleed — 1
   Cardiac arrhythmia — 1
   Spontaneous bacterial peritonitis — 1
2. Intraoperative death ($n = 1$)
   Arrhythmia and hypothermia — 1
3. Post-transplant deaths ($n = 4$)
   Small bowel infection — day 3
   Sepsis, embolic stroke — day 18
   Myocardial infarction — day 21
   Iatrogenic death — day 22

Table 7. Antiviral Agents That Can Be Used to Prevent Liver Allograft Infection with HBV

Acyclovir
Ganciclovir
Ara A
Ara AMP
Ribavirin
Thymosin α_1
GM–CSF

survival persisted. Although unsuccessful at the stated goal of preventing HBV disease recurrence, this study demonstrated several things. First, it demonstrated that IFN can be given to LTx recipients without adversely affecting allograft function. Second, it demonstrated that the use of IFN in LTx recipients does not initiate allograft rejection. Third, it demonstrated that IFN, although not successful in preventing disease recurrence, did prolong allograft and patient survival. This latter event may be a consequence of impaired viral replication during the early LTx period when large doses of immunosuppressives are being utilized.

Since this study was initiated, several other agents have been developed which can be added to the list of agents known to exist as treatment for HBV disease. These are shown in Table 7. The most promising of these appears to be thymosin α_1. Based upon preliminary evidence, it appears as if thymosin can induce clearance of HBV infection without any untoward effects.

Thus, based upon current knowledge, it is at least possible that one or more of these additional agents, if used either alone or in combination or even sequentially with either IFN or thymosin, may lead to an eradication of HBV in LTx recipients.

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The Authors

David H. Van Thiel, MD, is medical director of the Oklahoma Transplantation Institute (OTI) and director of the Hepatology and Digestive Disorder Center at Baptist Medical Center of Oklahoma (BMCO) in Oklahoma City.

Stefano Fagiuoli, MD, is a specialist in gastroenterology and hepatology, and transplant medicine at OTI.

Paolo Caraceni, MD, is a research fellow at OTI.

Harlan I. Wright, MD, is a specialist in gastroenterology and hepatology, and transplant medicine at OTI.

Judith S. Gavaler, PhD, is director of Women's Research and Informational Source at OTI.

Gunshot Wounds Traversing the Mediastinum: Guidelines for Evaluation and Management

Oscar Falcon, Jr., MD; Christopher J. Knott-Craig, MD; Ronald C. Elkins, MD

When there is suspicion that a penetrating wound to the chest has crossed the midline, it is incumbent upon the medical staff to actively exclude injuries to mediastinal structures such as the heart, aortic arch, bronchial tree, and esophagus, even though injury to such structures may not be immediately clinically apparent. The successful management of a patient with a gunshot wound apparently traversing the anterior mediastinum is presented; an esophagram identified an unsuspected through-and-through injury to the esophagus. The evaluation of this patient is discussed, and an algorithm for the evaluation and management of similar patients is proposed.

GUNSHOT WOUNDS TO THE CHEST ARE ASSOCIATED with high morbidity and mortality, particularly when the bullet traverses the mediastinum.[1-4] For a successful outcome, all major structures need to be systematically evaluated and appropriately treated.[5-8] Though this would seem to be common sense to experienced surgeons, caveats in the evaluation of such patients are not infrequent in institutions that have a low volume of major trauma cases.

In this manuscript, we review the management of a patient with a gunshot wound to the chest which traversed the midline. A penetrating injury to the esophagus was diagnosed, even though this was clinically thought unlikely based on the suspected trajectory of the missile. An algorithm which may be useful in the evaluation of similar patients is presented.

Direct correspondence to Christopher J. Knott-Craig, MD, Division of Thoracic Surgery, University of Oklahoma Health Sciences Center, Post Office Box 26901, Oklahoma City, OK 73190.

Case History

KG, a 23-year-old black male victim of a drive-by shooting, suffered a low-velocity gunshot wound to the chest. On arrival in the emergency room, he was fully conscious, dyspneic, and had a systolic blood pressure of 80 mmHg and a heart rate of 120/min. There was an entrance wound in the *left* shoulder but no exit wound. Mild subcutaneous emphysema was present bilaterally over the upper chest. Initial evaluation included a chest roentgenogram which showed bilateral hemopneumothoraces and a large caliber bullet lodged in the anterior chest wall just below the *right* clavicle. The mediastinum was reported as normal.

Bilateral chest tubes were placed, and initially drained 150 ml and 800 ml respectively from the left and right pleural cavities, without significant subsequent drainage. The patient was easily resuscitated with crytalloid volume replacement, with normalization of vital signs and respiratory status. An emergent aortic arch and four-vessel arteriogram to exclude a vascular injury, was normal (Fig. 1).

Based on the expected trajectory of the bullet—left anterior shoulder to right anterior chest wall—the bullet was thought to have crossed the anterior mediastinum and an esophageal injury was therefore considered very unlikely, particularly since the surgical emphysema was resolving, no significant air leak was present, and the general clinical status of the patient was excellent; on the basis of this assessment the patient was returned to the ward without having the contrast study of the esophagus which had

Figure 1. Arch aortogram and four vessel arteriogram of a patient with a gunshot wound to the left shoulder, with the bullet lodged below the right clavicle.

Figure 2. Gastrografin swallow showing leakage of contrast material into the right pleural cavity.

Figure 3. Gastrografin swallow on postoperative day 6 showing an intact repair.

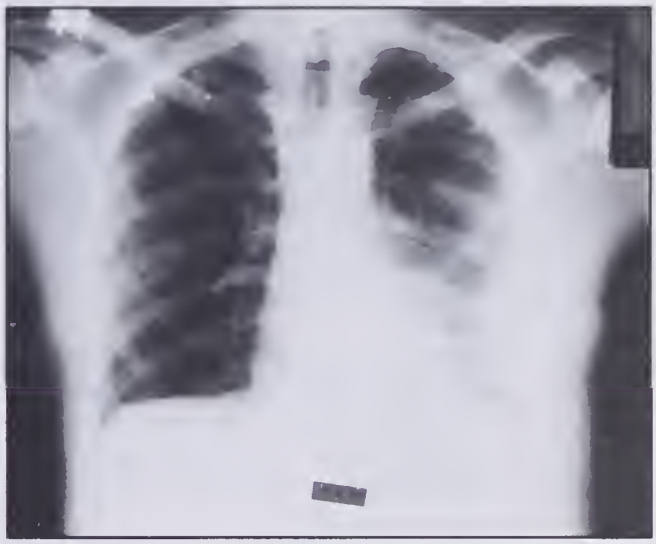

Figure 4. Predischarge chest x-ray showing good resolution of left empyema.

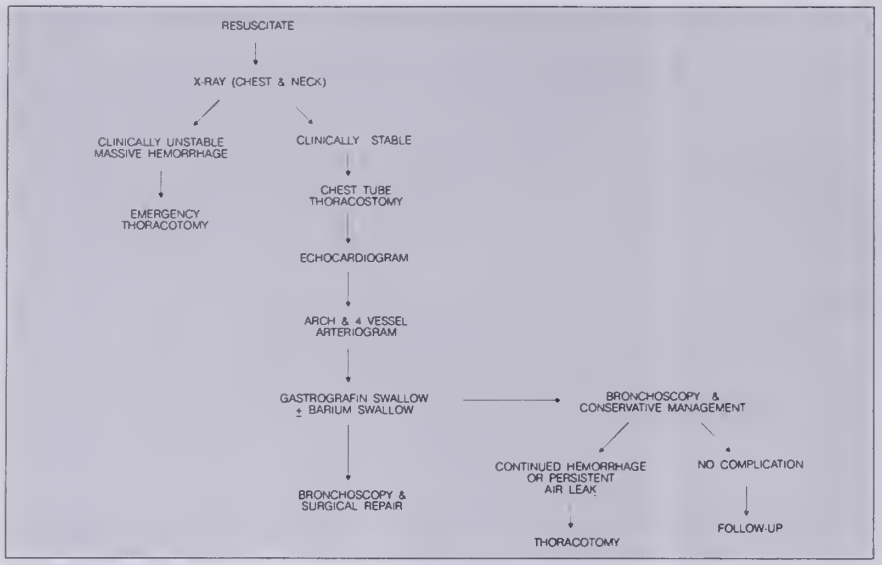

Table 1. Algorithm for the evaluation of patients with penetrating injuries traversing the mediastinum

been scheduled. At the insistence of the surgeons, however, the patient was returned to the radiology suite for a gastrografin swallow. This revealed extravasation into the right chest at the level of T-4 (Fig. 2), and the was patient taken to the operating room emergently. The time between the injury and surgery was now approximately 15 hours.

After induction of anesthesia, the trachea and proximal bronchial tree was examined bronchoscopically. Through a right posterolateral thoracotomy, the right upper lobe of the lung was repaired and the through-and-through gunshot wound of the esophagus was repaired in two layers. The esophagus was wrapped with a pedicled pleural flap to buttress the repair.[5] The mediastinum was widely opened on the right side and drained with an additional chest tube. Through a separate incision, a gastrostomy was performed and a jejunostomy feeding tube was placed.

A contrast swallow on the sixth postoperative day showed an intact repair (Fig. 3), and oral intake was commenced. Postoperatively, a left-sided empyema required surgical drainage. The patient was finally discharged 17 days after his injury (Fig. 4) and was asymptomatic six months later.

Comment

Successful management of penetrating chest injuries depends on a high index of suspicion and a clear strategy to identify possible injuries to all the major intrathoracic structures.[1,2,6,8] This management plan should be tempered by the clinical status of the patient[6-8]—a patient exsanguinating through a left chest tube needs immediate left thoracotomy and not numerous investigations, if he is to survive.

For the patient who is easily resuscitated and remains relatively stable clinically, as the patient presented in this report, the emergency room physician and surgeon need to have a clear and logical management plan designed to rapidly identify injuries to the major thoracic structures in order of priority. Such an algorithm is presented in Table 1 and enables the heart, great vessels, esophagus, upper airways, and finally the lung parenchyma and chest wall to be evaluated in that order. This should reduce the risk of overlooking an important injury that is not immediately apparent, thus benefiting the patient in terms of significant mortality and morbidity. In situations where the patient needs to be explored without delay, intra-operative bronchoscopy and esophagoscopy should be undertaken, and the missile tract carefully explored to exclude injuries to these structures. It is also important to realize that fiberoptic bronchoscopy performed after the patient has been intubated may fail to identify injuries to the proximal trachea—in this case the endotracheal tube needs to be temporarily withdrawn to the level of the larynx while the proximal trachea is being visualized. Alternatively, rigid bronchoscopy should be used.

It is important to realize that the pathway of the bullet is frequently erratic and the expected trajectory, as judged by entrance and exit wounds, may be misleading.[7] This is exemplified by our patient, in whom an esophageal injury was thought to be unlikely judging from the entrance wounds and the position of the bullet on the roentgenogram. In fact, initially an esophagogram was considered unnecessary. However, making the diagnosis of an esophageal perforation early is crucial, since the time from injury to repair correlates closely with mortality and morbidity in esophageal perforations.[1-6]

The high mortality associated with gunshot wounds to the chest which transverse the midline of the patient can be minimized only by a careful and logical approach to the patient, aided by a high in-

dex of suspicion. Because the associated mortality is often time related, this approach should be a well-integrated multidisciplinary effort involving emergency room physician, surgeon, radiologist, and anesthesiologist. The algorithm presented here represents a common-sense approach to the evaluation of such patients, and may help to ensure comprehensive evaluation and management of similar patients, particularly at times when the initial evaluation rests with the more junior members of the surgical team. J

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The Authors

Oscar Falcon, Jr., MD, is a general surgery resident at the University of Oklahoma Health Sciences Center (OUHSC).

Christopher J Knott-Craig, MD, is associate professor of surgery, Section of Thoracic Surgery and Cardiovascular Surgery, OUHSC.

Ronald C Elkins, MD, is professor of surgery and chief of thoracic and cardiovascular surgery, OUHSC.

SPECIAL

The Unspoken Chief Complaint: Family Violence

A Report from the OSMA Committee on Family Violence

Edward N. Brandt, Jr., MD, PhD, Chairman

THROUGH PUBLICATION OF ARTICLES SUCH AS "THE Battered Child Syndrome" by Kempe, et al,[1] physicians have learned that the family does not always provide the caring, loving atmosphere that we would hope. Since then, we have learned of the existence of other forms of family violence, namely spouse/partner abuse and elder abuse. Spouse/partner abuse includes violence towards one's current or former intimate partner, and elder abuse indicates physical or other abuse of elderly persons living in the same household.

To help counter what has become an epidemic of family or domestic violence, the American Medical Association has formed an Advisory Committee on Family Violence composed of representatives of various specialty societies. Two Oklahomans, Dr. Lori Hansen and Dr. Tisha Dowe Webb, serve on that committee. To ensure Oklahoma's involvement, presidents James Funnell and Lance Miller appointed an OSMA Committee on Family Violence chaired by Dr. Edward N. Brandt, Jr., and including as members Dr. Hansen, Dr. Webb, Dr. Richard Campbell, Dr. JoAnn Carpenter, Dr. Robert Block, Dr. David McCarty, Dr. Kenneth Muckala, Dr. Mary Saddoris, Ms. Susan Paddack, and Ms. Sherry Strebel. In addition, several experts also work with the committee, including Ms. Georgie Rasco of the Oklahoma Coalition on Domestic Violence and Sexual Assault, Ms. Ann Lowrance of the Department of Mental Health and Substance Abuse Services, and Dr. Karen Goldsteen of the University of Oklahoma Health Sciences Center. Finally, Ms. Anna Likos, a third-year medical student, works with the committee. The purpose of the committee is to increase Oklahoma physicians' awareness of this problem and of methods for helping people who are being abused.

Recognition and intervention are essential to break the cycle of violence.

What can physicians do? Most importantly, they can identify the signs and symptoms of abuse and ask about violence in the family. When the diagnosis has been made and treatment instituted, physicians must assure that community resources are brought to bear to help the abused. Recognition and intervention are essential to break the cycle of violence. The physician's role is to (1) recognize the presence of potential abuse; (2) treat the physical and psychological injuries; and (3) institute appropriate intervention to prevent future abuse.

Epidemiology

In America, according to the American Academy of Family Physicians:

❑ Abuse, child sexual abuse, and child neglect are reported each year;

Direct correspondence to Edward N. Brandt, Jr., MD, PhD, Co-Director, Center for Health Policy, University of Oklahoma Health Sciences Center, Post Office Box 26901, Oklahoma City, OK 73190.

❑ More than 1,000 children die each year from abuse and neglect;

❑ The single most common source of injury to women is violence perpetrated by their husband or boyfriend (this exceeds the number of women injured in car crashes, muggings, and rapes *combined*!);

❑ One-third of pregnant women suffer beatings by husbands or boyfriends;

❑ Nearly two million elderly are physically hurt or threatened by family members each year.

❑ This is not just a national problem, but also an Oklahoma problem. Consider the following:

❑ In 1992 Oklahoma police responded to 15,552 domestic abuse disturbances, according to the Oklahoma State Bureau of Investigations.

❑ In 1992, Oklahoma domestic violence shelters housed over 12,000 women and children, according to the Department of Mental Health and Substance Abuse Services.

❑ One out of every ten Oklahoma women surveyed stated they had been physically abused at least once during a pregnancy, according to the State Department of Health.

This is a scathing indictment of American family life. Physicians can and must be prepared to do something about it. The following information is intended to help them do so.

Physicians must become better informed in identifying the physical and emotional injuries from family violence and assume the responsibility of helping the victims.

Diagnosis

There are four principles to remember:

1. Abuse is characterized by denial. Examples of explanations of abusive injuries include the child "fell off the bed" or some other piece of furniture, a woman saying that she "ran into a wall" or "fell down the stairs," and similar superficially plausible stories. Therefore, it is essential that the physician ask directly about the cause of the injuries, and not accept an answer that does not fit the pattern of the injuries. It is also vital that such questioning take place in the absence of the potential abuser, since husbands or boyfriends may accompany the victim to ensure that she does not tell the truth. It is also important that questions be asked in a non-judgmental and non-threatening manner. The most important consideration is the current and future safety of the patient.

2. Chief complaints are often unrelated to violence. Children and adults may present with non-specific complaints such as sleeplessness, nightmares, depression, or anxiety. When such vague symptoms are given, it is vital to inquire about feelings of safety or specifically about violence.

3. Maintain a high suspicion index, recognizing that respected citizens, and indeed physicians, may be abusing members of their family. High socioeconomic or educational status does not preclude violence.

4. In cases of suspected abuse, immediate help must be offered and provided. This in itself may encourage the abuse victim to be more forthcoming, but the physician must follow through, or else the victim's sense of impotence is strengthened further.

The New York Governor's Commission on Domestic Violence and the AMA have developed some keys to diagnosis. They include:

The type of injury or extent of injuries *is inconsistent with the explanation given by the patient.* For example, cigarette burns, bite marks, fingernail scratches, etc., are rarely encountered except in domestic violence. Also, a central pattern of injuries such as to the head, neck, and chest are indications of violence.

Injuries *consistent with a defensive posture* such as forearm bruises or fractures.

Injuries *during pregnancy,* especially involving the breasts and abdomen.

The presence of *multiple injuries in various stages of healing* should suggest abuse of the child or adult.

Delay in seeking treatment in both children and women. There is often a wait of several days before seeking treatment either because they are prevented from doing so or because of their embarrassment.

Frequent visits for vague complaints without evidence of disease.

Alcohol abuse, drug use, or suicidal thoughts or attempts should lead to further questioning about abuse.

Abuse of children and spouses occur together commonly. Hence, if evidence of abuse of one is found, look for abuse of others.

Treatment and Intervention

Clearly, all injuries need treatment. However, it is

important to recognize that violence also results in psychological and emotional damage. Those effects must also be treated. Most importantly, the physician must reassure the patient that he or she does not deserve to be abused and is not alone. Community resources such as shelters must be utilized. The key words are concern, respect, and support. The victim must be made aware of the risk of recurrent abuse. Given the options—shelter, police intervention, or staying with friends or family—the physician must support the patient's choice even if not agreeing with it. Remember, all criminally inflicted injuries (including those resulting from family violence) must be reported immediately by the physician to law enforcement authorities, and the patient must be advised of that fact. Unfortunately, such reporting may increase the abuse and therefore, the patient must be made to recognize that, prepare for the consequences, and prepare to seek refuge.

Documentation of injuries via photos or a body map is very important even if legal action is not being currently pursued by the abused. This available evidence may be the catalyst for legal action against the abuser in the future.

Conclusion

Family violence is of epidemic proportions in the U.S. and in Oklahoma. Frequently, patients will come to physicians with vague complaints as an indirect way of seeking help and assistance out of their abusive situation. Oklahoma has an intricate system of 25 domestic violence programs and all offer 24-hour crisis intervention services to families besieged by intimate violence. The State Department of Mental Health and Substance Abuse Services operates a 24-hour statewide toll free hotline for referrals and assistance, 1-800-522-9054. Physicians must become better informed in identifying the physical and emotional injuries from family violence and assume the responsibility of helping the victims. The reader is referred to the bibliography for further assistance.

In closing, let us quote from a speech given by Dr. Robert McAfee, president-elect of the American Medical Association: "We the people of the United States of America, in order to form a more perfect union and to assure domestic tranquility, will allow more women to be killed in their homes by their husbands or boyfriends in a five-year period than Americans were killed in the Vietnam War! We the people!"

References

1. Kempe CH, Silverman FN, Steel BF, et al. The battered child syndrome, *JAMA* 1962;181:12-24.
2. Salber P. Domestic violence, *California Physician*, 1993.
3. Special issue on domestic violence, *JAMA*, June 17, 1992.
4. Diagnostic and Treatment Guidelines on Domestic Violence, Diagnostic and Treatment Guidelines on Child Abuse, Diagnostic and Treatment Guidelines on Elder Abuse, AMA

Additional Resources

1. Department of Mental Health and Substance Abuse Services, Ann Lowrance, Director of Domestic Violence and Sexual Assault Programs. (405) 271-8777, 1200 N.E. 13th, Oklahoma City, OK 73152-3277.
2. Oklahoma Coalition on Domestic Violence and Sexual Assault. Georgie Rasco, Administrator, (405) 557-1210, 2200 Classen Blvd., Suite 1300, Oklahoma City, OK 73106.

The Author

Edward N. Brandt, Jr., MD, chairs the OSMA Committee on Family Violence. He is also co-director of the Center for Health Policy Researach and Development and professor, department of health administration and policy, at the University of Oklahoma Health Sciences Center in Oklahoma City.

Look Out for Landmines

Hospital purchases of physician practices can be fraught with peril

Sandra Lee Breisch

The movement toward integrating physician and hospital services is still young enough that few of these complex relationships have had a chance to break up. But integrated delivery systems are running into problems—sometimes even before the ink has dried.

Frequently, conflicts arise because hospital purchases of physician practices are poorly structured or poorly understood by participants. But while the landmines may have been planted during the negotiations, they often don't explode until later.

"When the initial euphoria wears off, there's a coming back to earth," says Charlotte, N.C., attorney Keith Korenchuk. The expectations physicians had suddenly meet the reality of the system and their roles in it.

And for physicians who didn't go in with their eyes open, that can bring a major explosion, says Korenchuk, who has overseen several physician-hospital integration projects and is author of *Transforming the Delivery of Health Care: Mergers, Acquisitions and Physician-Hospital Organizations* (Aspen, 1993).

Agreeing on goals could be the key to resolution. But that can be difficult. Hospital managers are generally most interested in building patient flow, economizing, and showing a profit, while doctors tend to emphasize preserving independent medical decision-making to uphold their ethical obligations to provide the best care possible, regardless of cost.

And as sharply as those values might clash at the outset, Korenchuk warns that strain on the physician-hospital relationship will grow even more severe as the integrated system takes on more risk for the care it offers. "There will be pressure to remove practitioners who don't perform as well under a managed care environment."

Physicians who aren't cut out for a larger group practice will be inclined to leave the system because they won't be happy in it. "The second thing is credentialing and asking people to leave. I think we'll see more of that as the systems come under more economic risk."

But some of these problems can be avoided simply by knowing what they are and dealing with them up front. That might mean structuring an agreement that will minimize conflict. Or it could mean recognizing that practicing in an integrated system is not for you.

The following are some of the most common friction points between physicians and hospitals in integrated systems. Considering these potential landmines as you develop a system might help you avoid them.

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Work Expectations

Hospitals buying practices often assume that the selling physician will continue to see the same number of patients and generate the same number of referrals as before the sale. Hospitals "have learned the hard way that once the physician has signed the contract he or she is not on a 75-hour work week, but a 40-hour work week," says consultant Robert O. Graves, Graves & Co., Glendora, Calif.

The result is that hospital managers feel they overpaid for the practice or were cheated. Relations go downhill from there.

Solutions include explicitly outlining work hours in the sale agreement and structuring contracts so the physician and the hospital share risks and rewards.

Patient Retention

Practice volume also may drop because of patient loss after a sale. "Most of these patients are used to seeing their good old doctor, and when the hospitals take it over, usually they bring in various nomadic physicians. So the patients see somebody different every time they go in there, and they don't like it. They ask to have their records transferred to somebody else," says Roy Huntsman, a practice consultant with Medical & Dental Management Inc., Gainesville, Fla.

Solutions include retaining the selling physician to continue seeing patients or to transfer patients to a physician or physicians with long-term commitments to the system.

Referrals

Restrictions on referrals can be a clinical as well as a business problem. "Can physicians refer patients to a specialist he thinks is best or must he refer to a specialist who is a hospital employee?" asks Wheeler Chapman of Health Care Management Consultants, Northbrook, who's fought vigorously against hospital-employee integration. Referring only to hospital employees also can trap a physician, who might have a hard time getting referrals if he or she decided to leave the network.

Make sure any referral restrictions are clearly outlined and acceptable before signing a contract.

Retirement

Once you go to work for a hospital, you lose the ability to fund your own pension plan and must rely on what the hospital provides. Understand the financial consequences before you commit.

Treatment Protocols

Who decides how patients should be treated and by whom can be a major friction point. Hospitals may want to control the protocol to save money, but physicians are still liable for their actions.

"Medical decision is always first," says Chapman. "There's a California malpractice case where the doctor failed to perform services for an HMO patient because they weren't covered under his contract. The judge said, 'Tough luck, doctor, the patient always comes first.'"

The only real solution is to insist that physicians have the option to exercise their own judgment at any point, says Ken Bowben of Berkshire Professional Management Inc. in Pittsfield, Mass. "All physicians subscribe to a code of ethics, and every contract I've ever seen always makes the corporate rule subservient to the code of ethics, canons of professional conduct. I certainly wouldn't have recognized medical decisions come first."

Staffing Issues

Doctors often are very loyal to their staffs and want their jobs protected when they sell their practice. But one, thing hospitals usually have in mind when buying practices is to streamline office procedures, often by combining administrative functions from several practices and reducing staff.

Further complicating the problem can be cultural and business differences between private physician offices and hospitals. Employees who may be comfortable with a small office may not like the bureaucracy of a large institution. Practice employees' fitting into a hospital union also can be a problem.

Loss of staff can lead to loss of patients, Huntsman notes. "The patients get further discouraged because not only are they losing their doctor, but their nurse or the person they've been talking to on the phone for 30 years."

If you want to keep your staff, be sure the deal is set up that way.

Payment Policy

Hospital payment and collection policies tend to be stricter than physicians', Huntsman says. That also can discourage patients, who might avoid seeing the doctor for fear of being harassed about the money.

Once you are in an integrated system you will probably lose control of billing. Make sure you're comfortable with the new rules.

Evaluations

As a hospital or network employee, you may be subject to employee evaluations beyond the peer review you're used to. And that may be a problem, Bowben says. "Hospitals don't know how to evaluate physicians. That's not a rap on them, it's just not their traditional function."

Before signing a contract, you may want to spell out who will be doing evaluations and on what basis.

Incentives and Bonuses

Related to problems with evaluations are problems with bonuses. "If you don't know what the doctors are going to produce, how are you going to measure performance above or below? The hospital doesn't have the capacity to measure productivity. They don't know what the rules are," Bowben points out.

Goodwill or Salary?

Often hospitals buying practices will pay up front for goodwill—the practice's value beyond the worth of its hard assets. But often, goodwill payments will be parceled out over several years.

The problem arises when it isn't clear what the hospital is paying for salary and what it's paying for goodwill. For example, after five years a hospital might cut your salary on the theory that your compensation was artificially increased to compensate for goodwill, and now the goodwill has been paid for.

The solution is to be very clear about what the terms of your compensation are and how much you are being paid for your practice.

Loss of Autonomy

For a physician who has been in control of every aspect of his or her practice for years, the loss of autonomy that comes with becoming a hospital employee can be profound. "The hospital is in control of your destiny. And the hospital's goal is to control medicine and have the doctors as employees," Chapman says.

If this bothers you, the solution is not to sell your practice unless you're ready to leave, Huntsman says. "I think most physicians are smart enough to know that if they're going to sell their practice and work for a big corporation like a hospital, they're going to lose a lot of their personal freedom and they just don't do it. They do it to retire, but not to continue practicing."

Peer to Employee

Along with loss of autonomy comes a shift in status from an independent peer of the hospital to an employee. "This is an amazing change and the physicians we've worked with have been pretty well shocked taking orders from someone they've been peers with before," says Stevan D. Olian, president of PMC-Consultants to Health Professionals in Burlingame, Calif.

Termination Clauses

Together with compensation and buyout terms, termination clauses are the most important features of an integration deal, says Robert Krypel, president of Northbrook, Ill.-based Health Care Management Group of America and a member of the AMA's Managed Care Advisory Board. That's because practice sales usually include some kind of noncompete agreement to prevent the physician from selling out, then turning around and setting up a new practice to serve existing patients. But the noncompete agreement also would prevent the doctor from reestablishing a practice if he or she were terminated.

To protect against this risk, termination conditions should be specified in the practice sale contract. "I suggest that the only basis for termination should be cause, defined as, but not limited to, loss of license, loss of staff privileges or the commission of a felony," Krypel says.

"If they can only be terminated for cause, then they do not run the risk of their employer deciding to replace them with the lower-paid physician."

You also might negotiate to repurchase your practice at a given point, says Olian.

Office Location

Once a hospital has bought several practices, it will often try to save money by consolidating them into fewer facilities. Problems range from losing your staff and office to extending your commute.

One way to solve the problem is to require your office to stay open in its present location as part of the purchase agreement. This also may help you hold on to staff.

Liability Tail Coverage

Who pays for professional liability tail coverage can be a contentious issue when you get ready to leave an integrated network. If you have to pay, it creates a substantial penalty for leaving. Make sure the agreement is clear on who pays.

Cost Shifting

Physicians who sell their practices to hospitals often find themselves the victim of hospital accounting practices, Olian says. While you may expect to continue netting a certain income based on your historical charge volume and overhead costs, you may find that your profits are being eaten up by cost-shifting.

"It's often called dean's costs or administrative tax, and sometimes the rent is increased. A thousand things can happen, where the ground can be shifted underneath them. So the contracts have to be very specific, particularly if it is a profit-incentive or profit-sharing contract," Olian says.

Payment Basis

Many physician employees are paid in part on how much revenue they generate But the physician has no control over crucial functions influencing revenue. If the hospital' s collection function is poor or if the network signs low-reimbursement contracts, you may find yourself working harder for less, Olian says.

"Another thing in fine print is they have hidden phrases that allow them to increase or decrease salaries dependent upon whether the hospital is making money," Olian notes. One hospital system he works with last year lowered medical staff salaries 5% across the board because of declining profits. "It's in the contract; they have the right to do it."

The best way to avoid misunderstandings about payment rates is to thoroughly understand the contract before you sign.

Unexpected Administrative Tasks

"Many contracts, in the fine print, require that as employees [physicians] perform other administrative work, like train residents, handle other departmental paperwork and so forth," Olian says. Again, the key here is to read and understand the contract before you sign.

The Author
Sandra Lee Breisch is a free-lance writer based in Chicago.

COMMENTARY

We ophthalmologists are sinking lower and lower, fighting each other, back-biting, and belittling each other.

Whores Are Us

Thomas O. Nicklas, MD

SOMEONE ONCE SAID: "ALL THAT IS NECESSARY for the forces of evil to win in the world is for enough good men to do nothing."

In 1968, when I finished my training, my partner and I moved to Durango, Colorado, to establish an office practice in ophthalmology. At that time, ethical strictures permitted the office sign to have only 2½ inch letters, and the newspaper ad announcing the opening of our practice was restricted to 2 × 3 inches with only our names on it announcing the opening. The ad was permitted to run for only three days, and if it ran longer we would be subject to ethical criticism from our fellow physicians. We did not mind these strictures, as they applied to all physicians, and the patients began to come in anyway.

Twenty years later, in Hawaii, I heard a cataract surgeon talk to a large group of young ophthalmologists about "internal and external" marketing. He told about radio and TV advertising. He told of flying to various islands, setting up a tent to give free cataract and glaucoma examinations—and drinks and snacks if the patients were senior citizens. He strutted around because he was a "high volume eye surgeon" that everyone wanted to be like. He had an entourage of aides and assistants that catered to his every need; it was rumored that he worked from 8:00 AM to 4:00 PM just to meet his overhead. He advised sending the patients flowers and mementoes and pictures of the patient with the eye patch still on posing with the doctor.

Now some of the high volume ophthalmologists send optometrists out in vans to distant areas to give free examinations to search for cataracts and other surgical problems to send to the surgeon. There is undue emphasis on "laser surgery," or on "three stitch," "two stitch," "one stitch," or "no stitch" surgery, with an implication that it is only available from one surgeon. There are so-called support groups for post-surgical get-togethers, barbecues, and "stare and share" groups. (I can see better with *my* implant than you can!) Many high volume surgeons send out periodical newsletters detailing missionary work, lectures, and other professional doings "for the sake of the masses," so that an altruistic image is projected.

We ophthalmologists have broken every ethical rule that was ever written by our forefathers regarding advertising and patient care. We have started a trend of consorting with the optometrists that will destroy ophthalmology and join us to the optometric society. Optometrists have already tried to pass a legislative bill that would make them the official primary care physicians for all eye care, with control of all eye referrals. It is frightening that such a bill was even considered, and it is sobering to realize that optometrists outnumber ophthalmologists, and they work hard at influencing legislators.

Recently I was called by one of the local optometrists who occasionally sends me a cataract patient when the patient refuses to go out of town. The optometrist said he wanted to do all of his patient's post-op care following the surgery. I explained to him that I was not comfortable with that and besides, post-op care is included in my surgery charges for three months. He said: "If you can code your surgeries with such-and-such number, I can do post-op care and receive $100 from Medicare." I told him that was fee splitting and that I had never done that before and since I

Direct correspondence to Thomas O. Nicklas, MD, Post Office Box 516, Grove, OK 74344.

knew what I had done in surgery, I would know what possible problems to look for that might arise after surgery: Did I break any zonules? Was there a small rent in the capsule? Did the patient bleed? Are there RBC, WBC, etc? Was the intraocular pressure up? Was extra viscoat left behind? He said: "Everybody else is doing it. You're the only one who is not doing it." When I told him again that I was not comfortable with it, he replied: "...then I'm not going to send you any more patients."

An intraocular lens salesman told me that one ophthalmologist tells the optometrists that if they are uncomfortable with the post-op care that they can keep the money that Medicare sends and he will do the post-op care anyway. Many ophthalmologists in big hospitals are scrambling to teach, entertain, and feed the optometrists at monthly "Grand Rounds" meetings. Some of the physicians I see doing this are men whom I highly respect for their expertise in ophthalmology, i.e., retina, cornea, glaucoma, etc., but they have been trapped in a fear of losing optometrists' referrals.

We ophthalmologists are sinking lower and lower, fighting each other, back-biting, and belittling each other. We are like a pack of wild dogs tearing each other to pieces, while the optometrists are gathering the funds and the political clout to carry out their legislative agenda.

In the late 1800s, there were over seven million buffalo in vast herds on the plains. They dwindled to less than 600 because of their indifference to each other's plight as the hunters methodically killed off the buffalo at the periphery of the herd. Stalin and Lenin once said they did not need to fight the USA in war because it would become rotten from within and crumble. We ophthalmologists are at a weakened state, and we are decaying from within. We must become better, and do better.

I once saw a movie with George C. Scott where he played a man in love with a prostitute. They were together one night and she was crying because she loved him but she was a prostitute. He said, "Honey, a whore is not a whore that sleeps with a man, but a whore is a whore who has the heart of a whore."

I believe that most of us ophthalmologists do not have the hearts of whores but instead we are doing it because the other guy is doing it. Let's clean up our act, stand and band together, and get our integrity and credibility back for ourselves. We need to set up some ethical standards and guidelines and live by them. If we allow greed, pride, and jealousy to continue to drive us apart, we will soon be like the extinct buffalo, standing alone in a national park to be observed by the laughing hyenas.

The Author

Thomas O. Nicklas, MD, has a private ophthalmology practice in Grove, Oklahoma, where he has been practicing since 1985.

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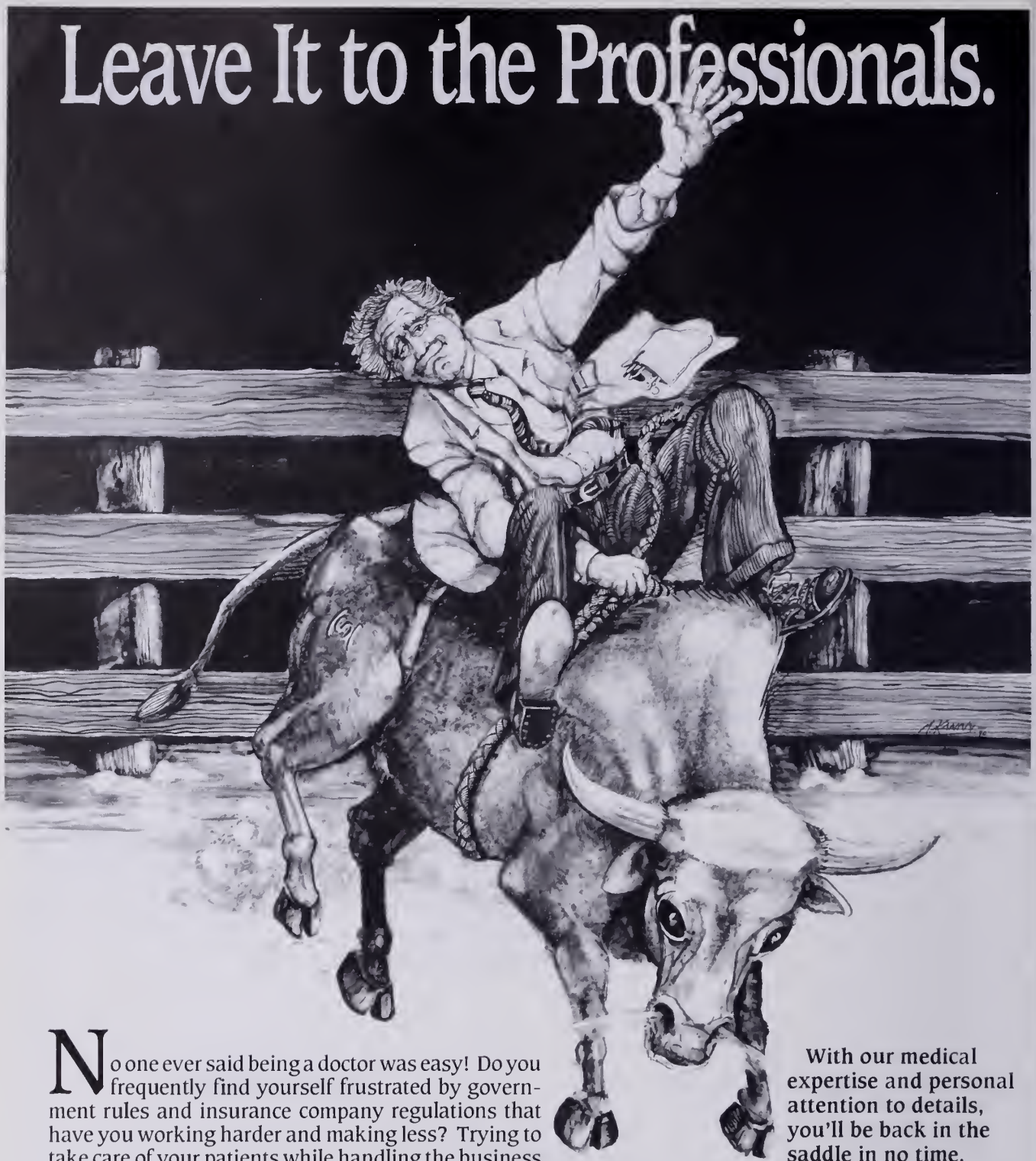

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Muskogee's Jay Gregory becomes 89th OSMA president

Dr. Jay A. Gregory

Jay Allen Gregory, MD, Muskogee surgeon, assumes the presidency of the Oklahoma State Medical Association this month during the Annual Meeting of the OSMA House of Delegates. He will be installed during a gala banquet Saturday, April 16, at Shangri-La Resort, becoming the association's 89th president. He succeeds Dr. G. Lance Miller of Tulsa.

Dr. Gregory is currently chairman of the Oklahoma Rural Health Association and chairman of the OSMA's Rural Health Council. He served as vice-chairman of the association's Board of Trustees from 1990 to 1992, and this year completes his term as chairman of the board.

He has been an alternate delegate to the AMA since 1989 and is a member of both the AMA's Practice Parameters Forum and its Advisory Panel for the Evaluation of Practice Parameters.

Dr. Gregory also assumes the presidency of the Oklahoma Chapter of the American College of Surgeons (ACS) this year. He was state councilor to the ACS from 1989 to 1992 and this year is a member of the college's Committee of 100.

Born in Alva, Dr. Gregory was raised and educated in Oklahoma. After earning an MS degree in medical microbiology at the University of Oklahoma, he attended the OU College of Medicine, graduating in 1976. He received his postgraduate training at the University of New Mexico School of Medicine in Albuquerque, where he also completed a fellowship in burn and trauma.

Dr. Gregory and his wife, Edith, have one child, Elijah.

Final vote on MCO/IPA awaits delegates at Shangri-La

At its February 27 meeting in Oklahoma City, the OSMA Board of Trustees approved six motions regarding the OSMA's proposed managed care organization (MCO) and independent practice association (IPA). The motions were then forwarded to the OSMA House of Delegates, which met later that day in a special called session.

In a meeting lasting approximately three hours, the delegates heard a report on the Garvey Group's preliminary survey and feasibility study, after which the board's motions were discussed at length, partially amended, and ultimately approved.

In essence, the motions provide for

(1) delaying the decision on how to capitalize the IPA and MCO until the April meeting of the House of Delegates and completion of the Garvey Group's feasibility study;

(2) completion of Phase II of the Garvey Group's feasibility study;

(3) authorization of expenditures necessary to complete the legal, financial, and organizational documents for the proposed IPA and MCO for submission to the House for a final vote of approval in April;

(4) approval of the board's recommendation that Oklahoma osteopathic physicians who are members of the Oklahoma Osteopathic Association or the American Medical Association be invited to participate in the proposed IPA and proposed MCO; and

authorization of the Board of Trustees to procede with but not implement the formation of (5) an IPA and (6) an MCO, so that final plans for both can be brought before the House of Delegates in April for approval. (Two interim boards of incorporators,

(continued)

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MCO/IPA decision *(continued)*

headed by OSMA President G. Lance Miller, MD, and President-Elect Jay A. Gregory, MD, have been appointed to work out the details of the proposals for House consideration).

Tom Garvey reported that his group's preliminary findings were based on 981 responses to survey questionnaires mailed via first class to all active members of the OSMA and the Oklahoma Osteopathic Association. Of those returns, 815 were from MDs and 146 from DOs. Seventy-nine percent of the respondents reacted favorably to the idea of a physician owned and controlled health system (27% Excellent, 20% Very Good, 23% Good), a percentage comparable to that found in similar surveys in other states, Garvey said. Personal interviews with business and community leaders around the state found employers to be generally interested and willing to cooperate.

Based on the findings to date, Garvey said, his group will probably recommend stock options rather than assessments as the way to capitalize the venture, with some $10 to $15 million, or $3,000 to $4,000 per physician, needed to get started. The OSMA House of Delegates will decide this month which funding method will be used.

The survey explored physicians' attitudes toward and interest in the concept of a statewide physician owned and controlled system, membership in the system, type of funding preferred (assessment vs. stock option), willingness to invest certain amounts, current systems in the state and level of satisfaction with them, and practice values and considerations such as gatekeeper systems, practice parameters, and peer review.

Life Memberships approved in February by Board of Trustees

Meeting in Oklahoma City on February 27, the OSMA Board of Trustees approved Life Membership applications from the following physicians:

Eugene Bell, MD, Tishomingo; Joan K. Leavitt, MD, J. Hartwell Dunn, MD, Arthur E. Schmidt, MD, and Clarence Shields, Jr., MD, Oklahoma City; Arthur F. Hoge, Jr., MD, Ketchum; William G. Husband, Jr., MD, Elk City; Charles R. Johnson, MD, Stillwater; and William P. Jolly, MD, Lawton.

Also: Melvin R. Swafford, MD, James E. White, MD, and James E. Kraft, MD, Tulsa; Winfred L. Medcalf, MD, Ardmore; and Edwin Pointer, MD, Tahlequah.

'94 Annual Meeting schedule

Dr. Robert McAfee

As this issue of the JOURNAL goes to press, the program below is the latest available information for the 88th Annual Meeting of the OSMA House of Delegates being held this month at Shangri-La Resort.

Robert E. McAfee, MD, president-elect of the American Medical Association, will be this year's keynote speaker. He will address a joint OSMA/OSMAA luncheon on Friday, April 15.

Thursday, April 14

11:00 AM OSMA Executive Committee
1:30 PM OSMA Board of Trustees

Friday, April 15

7:30 AM OSMA Hospital Medical Staff Section Breakfast
9:00 AM OSMA House of Delegates Opening Session
11:00 AM MCO/IPA presentation—by The Garvey Group
12:30 PM OSMA/OSMAA Luncheon Keynote Speaker: AMA President-Elect Robert E. McAfee, MD
1:30 PM OU College of Medicine Scientific Program
1:30 PM OSMA Physician Pension Plan Program—presented by OSMA Council on Member Services
2:00 PM OSMA Reference Committees
6:30 PM OU College of Medicine Alumni Association Reception and Dinner—Presentation of Physician of the Year and Amicus Medicinae Awards
8:30 PM AMA-ERF Western Party and Basket Auction

Saturday, April 16

7:30 AM OSMA Council on Rural Health
8:00 AM OSMA Past Presidents' and County Presidents' Breakfast
8:30 AM PLICO Loss Prevention Seminar
9:00 AM "Meeting the Challenge of Health System Reform" Seminar—presented by Harrison Associates, Inc.
10:00 AM "Medicare 1994" Seminar—presented by Harrison Associates, Inc.
10:15 AM Candidates' Forum
Noon OSMA Golf Tournament (4-person scramble)
6:00 PM OSMA/OSMAA Presidents' Reception
7:00 PM OSMA/OSMAA Presidents' Inaugural Banquet (Black tie optional)

Sunday, April 17

9:00 AM OSMA House of Delegates Closing Session
11:00 AM PLICO Forum (immediately following adjournment of OSMA House of Delegates)

IN MEMORIAM

1993

Maud Staveley-O'Carroll, MD	January 16
David Lloyd Bell, MD	January 23
William Ray Turnbow, MD	February 28
Waldo B. Newell, Jr., MD	February 16
Homer Clark Wheeler, MD	February 18
William Alfred Waters, MD	March 19
William Niebuhr Weaver, MD	March 20
Willie George Long, MD	March 22
William Finis Ewing, Jr., MD	March 31
John Robert McInnis, MD	March 31
Chester Randall Seba, MD	April 4
Edward Cotter Murray, MD	April 9
Eric McLain White, MD	April 23
John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13

Exotic pets and rabies: A combination that's hard to beat

In the past few years, it has come to the attention of the Communicable Disease Division (CDD) of the Oklahoma State Department of Health (OSDH) that many people think exotic animals (such as ferrets, monkeys, and pot-bellied pigs) make good family pets. Because of increasing interest in these exotic animals, physicians caring for anyone owning or considering owning one of these animals needs to be aware of the following:

1. Unlike dogs or cats, there is no licensed rabies vaccination for exotic animals (other than ferrets) recognized by the National Association of State Public Health Veterinarians. Therefore, exotic animals (other than ferrets) cannot be effectively immunized against rabies.

2. The period during which a dog or cat is able to transmit rabies and begins showing symptoms of rabies is usually 3 to 5 days. This time period in exotic animals (including ferrets) has not been researched and is unknown. Therefore, quarantine is not an option in the event an exotic animal bites someone (i.e., the person bitten could develop symptoms of rabies before the exotic animal does).

3. Because there are effective, licensed vaccines for dogs and cats, and a known time period between becoming infective and showing symptoms of rabies, dogs and cats are usually quarantined for a 10-day period when they bite a human, to ensure that rabies is not present. Unfortunately, exotic animals cannot be treated in the same manner. Because of the lack of a licensed vaccine, an unknown amount of time between the exotic animal becoming infective and showing signs of rabies, and the inability to interpret reliably signs of rabies, if an exotic animal bites someone, it may be necessary to euthanize and test the animal for rabies to avoid the necessity of the bitten person receiving rabies prophylaxis.

4. Exotic pets can carry exotic disease. For ex-

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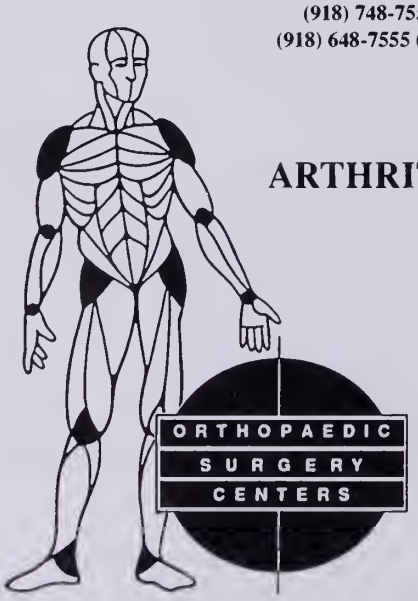

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LETTERS

Write on, Dr. Miller

I would like to express my appreciation to Dr. Lance Miller for his President's Page in the February issue of the JOURNAL. The title ["The Write Thing"] was clever and the message refreshing. While I, like most of our colleagues, am aware that the environment in which we practice is becoming increasingly complex, and our frustration level increased, it it important that our leadership remain positive. When confronted by many forces beyond our control, there is a tendency to become reactive, alarmist, and negative. Too often the statements from our professional organizations reflect a sense of desperation, and doom-saying is the way we respond. Such a defensive posture certainly does not enhance our public image.

Dr. Miller reminds us quite eloquently that ours is still a wonderful and satisfying profession as we practice it in Oklahoma, and that with the unity and mutual support which have characterized us in the past, we can keep it that way. This is an important concept not only for those of us practicing now, but for physicians in training and potential physicians to hear. Thank you, Dr. Miller, for encouraging us to maintain a healthy perspective.

—William O. Smith, Jr., MD
Claremore

Exotic pets *(continued)*

ample: Simian B virus is carried by certain types of monkeys.

The CDD asks that this information regarding exotic animals be discussed with and considered by patients before incurring the expense of owning one of these animals. Also, physicians caring for patients with exotic pets need to be aware of the special health hazards associated with these animals.

CAPSULES

■ **Frank G. Gatchell, MD, retired surgeon of the** Oklahoma City Clinic, has been named president of the International Mayo Alumni Association. The association consists of 11,000 members in 60 countries. Dr. Gatchell joined the Oklahoma City Clinic in 1957 after completing a four-year residency program at the Mayo Clinic. He retired from his surgery practice in 1990.

■ **Oklahoma City pediatrician Jane E. Puls,** MD, has been appointed associate councilor for the State of Oklahoma by the Southern Medical Association. As associate councilor, Dr. Puls will assist state councilor Charles A. Farmer, Jr., MD, in promoting membership in the association and encouraging participation in the many educational, professional, and practice-related programs and services offered by the association. Dr. Puls is affiliated with Children's Hospital of Oklahoma at the University Hospitals in Oklahoma City.

■ **Guidelines stating specific periods of disabil**ity for nearly 1,000 common illnesses, injuries, and medical procedures have been released in the second edition of *The Medical Disability Advisor: Workplace Guidelines for Disability Duration.* The 946-page book is designed as a tool for employers, insurers, and health care providers to manage disability claims and control disability costs. Recommended by the American College of Occupational and Environmental Medicine (ACOEM), it provides users with comprehensive

(continued)

CAPSULES (CONTINUED)

descriptions of approximately 700 diagnoses and 225 medical/surgical procedures and includes disability duration tables based on each of the U.S. Department of Labor's five job classifications. For information, contact ACOEM, 55 West Seegers Road, Arlington Heights, IL 60005, (708) 228-6850.

■ **Obstetrics and gynecology remains the highest** risk specialty for both liability losses and number of claims, reports the March *PLICO News*. The specialty has incurred some $25 million in losses since PLICO was established in 1980. General surgery was next, with $16 million, followed by family practice, $15 million, and anesthesiology, $13 million. The average loss per claim for all specialties was $30,667.

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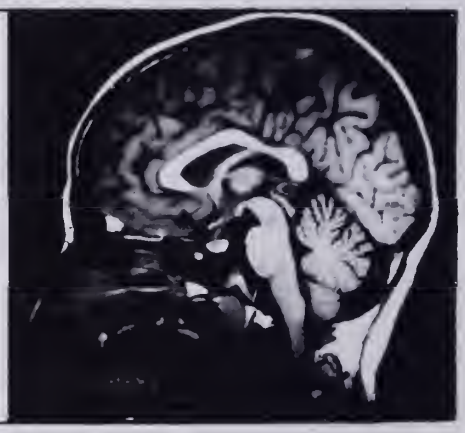

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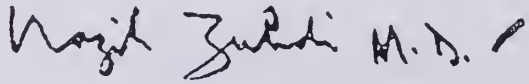

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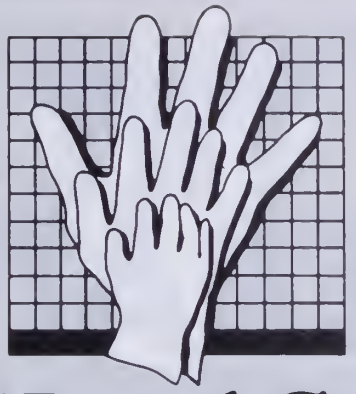

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THE LAST WORD

■ **D.J. Flournoy, PhD, has been named recipient** of the Charlotte S. Leebron Award for best scientific paper published in the JOURNAL in 1993.The JOURNAL's Editorial Board accorded Dr. Flournoy this special recognition not only for his paper "Incidence of Bacteria from Clean-Catch and Catheterized Urine of Nursing Home Residents," published in November, but also for his co-authorship of a second paper last year and his consistent submission of high quality papers for a period of 15 years. Since 1979 the JOURNAL has published a total of 23 papers authored or co-authored by Dr. Flournoy. He will be presented the $500 award during the Opening Session of this month's OSMA Annual Meeting.

Also being honored by the Editorial Board is Robert M. Smith, MD, Oklahoma City. Dr. Smith, contributor of several cover photos this year, will receive the Best Cover Photograph Award for his Red Rock Canyon photo, published in October 1993.

Dr. Flournoy is director of microbiology at the Veterans Affairs Medical Center and professor of pathology at the University of Oklahoma Health Sciences Center. Dr. Smith is an internist specializing in cardiovascular disease.

■ **The American Medical Association's position** on universal coverage and professional liability reform was detailed by Executive Vice-President James S. Todd, MD, in a March 2 letter to the executive directors of all state medical associations.

Regarding liability reform, Dr. Todd said the AMA specifically recommends (1) a $250,000 cap on noneconomic damages (recognizing that several states have a lower threshold); (2) mandatory periodic payment of future elements of damages; (3) mandatory offset of collateral sources such as health insurance and disability benefits when computing compensation to prevent double recovery of damages; (4) a sliding scale limit on attorneys' fees in relation to the size of the award; (5) a statute of limitations, applicable to adults and minors, to limit the time period for filing claims; (6) a certificate of merit as a prelude to filing medical liability cases and adopting basic criteria for medical expert witnesses; (7) encouragement of patient safety issues as an integral component of outcome and quality assessment programs; and (8) allowing providers who follow clinically relevant practice parameters developed by professional associations to raise such compliance as an affirmative defense in liability actions.

On universal coverage, the letter states, "Health care coverage must be extended to all Americans. We support a variety of approaches to achieve this goal: an employer mandate, an individual mandate, and health IRAs. As the congressional debate unfolds, flexibility will be needed in determining the relative responsibilities of individuals, employers, and government to ensure universal coverage with a standard set of health care benefits for all Americans."

■ **Some of the best doctors in the country are** members of the OSMA and practice at the University of Oklahoma Health Sciences Center, according to the second edition of *The Best Doctors in America*. Published by Woodward/White, Inc., the book is based on a year-long survey involving more than 13,000 telephone calls and 11,000 letters to health care professionals across the nation. The directory includes 7,200 physicians in more than 350 areas of medical expertise, or slightly more than 2% of the nation's 350,000 practicing physicians.

Listed are Drs. Harvey J. Blumenthal; James R. Couch, Jr.; Mark Allen Everett; Bradley K. Farris; John I. Fishburne, Jr.; William A. Grana; Jack V. Hough; James H. Little; D. Robert McCaffree; Michael McGee; Jesus E. Medina; Betty Pfefferbaum; Sanford Schneider; Gregory L. Skuta; Roger E. Wehrs; and Dennis A. Weigand.

■ **Medication errors are the second most frequent** and second most expensive causes of professional liability loss cited in a study of 90,000 claims nationwide by the Physician Insurers Association of America. The most common medication errors were incorrect or inappropriate dosage (more often the result of inappropriate frequency rather than strength of dosage), medication inappropriate for the medical condition, failure to monitor side effects, and failure of communication between physician and patient.

■ **Several more elective positions unexpectedly** will be open at the OSMA Annual Meeting this month. M. Joe Crosthwait, MD, chair of the Oklahoma delegation to the American Medical Association and a delegate for 20 years, has opted not to seek reelection as a delegate this year. In addition, Drs. Eugene G. Feild and James B. Eskridge III have announced they will not seek reelection to the PLICO Board of Directors.

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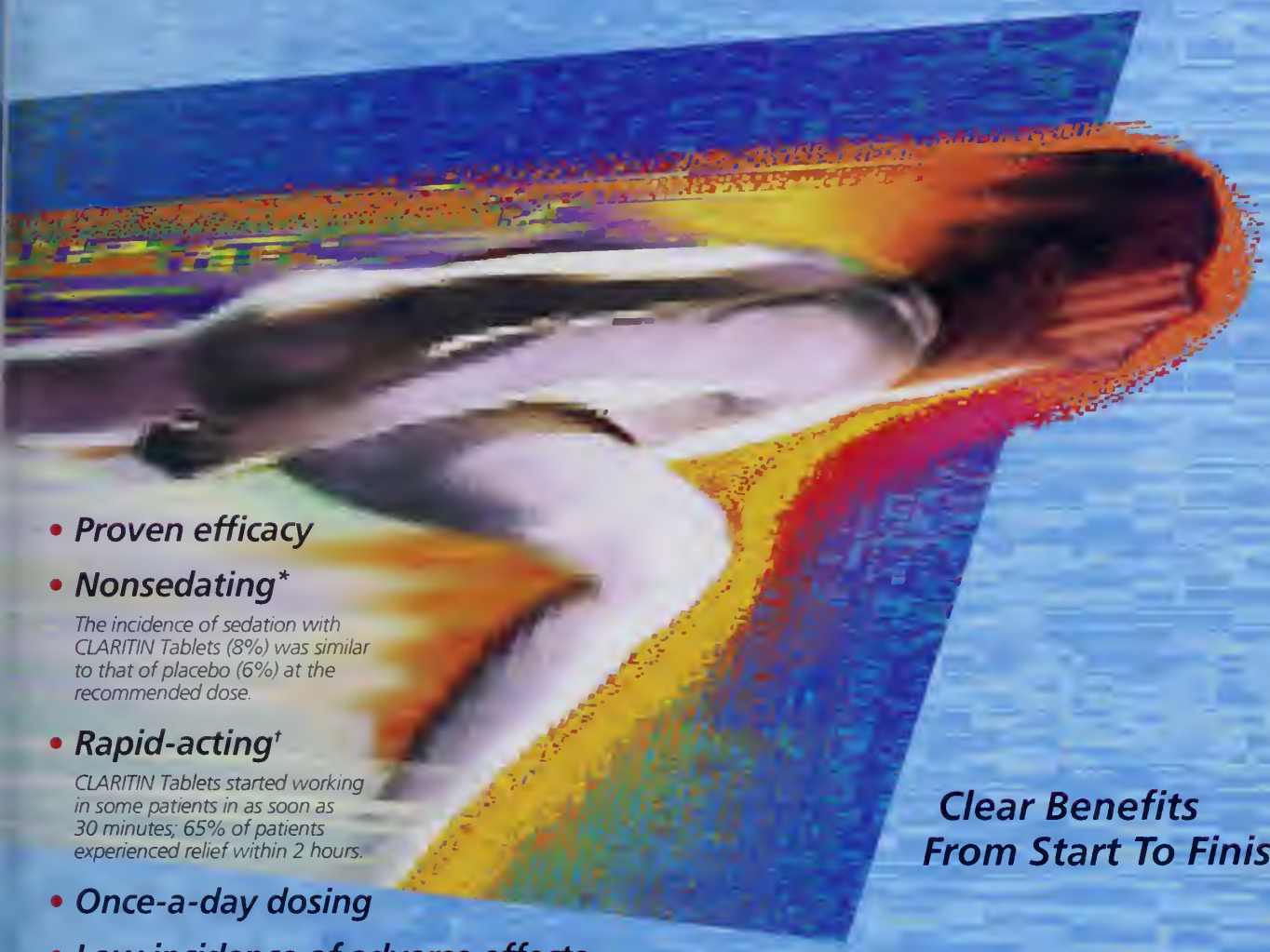

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- **Low incidence of adverse effects**

  In controlled clinical trials using the recommended dose, the incidence of headache (12%), somnolence (8%), fatigue (4%), and dry mouth (3%) with CLARITIN Tablets was similar to that of placebo (11%, 6%, 3%, and 2%, respectively).

- **Over 1 billion patient days of worldwide experience**

Clear Benefits From Start To Finish

Once-a-day

Claritin®

10 mg TABLETS (loratadine)

* In studies with CLARITIN Tablets at doses 2 to 4 times higher than the recommended dose of 10 mg, a dose-related increase in the incidence of somnolence was observed.

† Relief began in 13% of treated patients vs 4% of placebo-treated patients within 30 minutes (P=.04). At 2 hours, 48% of patients receiving placebo experienced relief. Distribution of onset times was significantly earlier for CLARITIN Tablets vs placebo (P=.03).

Please see following page for brief summary of Prescribing Information.

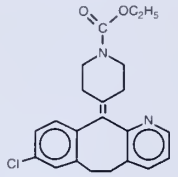
CLARITIN®
brand of loratadine

PRODUCT INFORMATION

TABLETS
Long-Acting Antihistamine

DESCRIPTION CLARITIN Tablets contain 10 mg micronized loratadine, an antihistamine, to be administered orally. They also contain the following inactive ingredients: corn starch, lactose, and magnesium stearate.

Loratadine is a white to off-white powder not soluble in water, but very soluble in acetone, alcohol, and chloroform. It has a molecular weight of 382.89, and empirical formula of $C_{22}H_{23}ClN_2D_2$; its chemical name is ethyl 4-(8-chloro-5,6-dihydro-11*H*-benzo[5,6]cyclohepta[1,2-*b*]pyridin-11-ylidene)-1-piperidinecarboxylate and has the following structural formula:



CLINICAL PHARMACOLOGY Loratadine is a long-acting tricyclic antihistamine with selective peripheral histamine H_1-receptor antagonistic activity.

Human histamine skin wheal studies following single and repeated 10 mg oral doses of CLARITIN Tablets have shown that the drug exhibits an antihistaminic effect beginning within 1 to 3 hours, reaching a maximum at 8 to 12 hours and lasting in excess of 24 hours. There was no evidence of tolerance to this effect after 28 days of dosing with CLARITIN Tablets.

Pharmacokinetic studies following single and multiple oral doses of loratadine in 115 volunteers showed that loratadine is rapidly absorbed and extensively metabolized to an active metabolite (descarboethoxyloratadine). The specific enzyme systems responsible for metabolism have not been identified. Approximately 80% of the total dose administered can be found equally distributed between urine and feces in the form of metabolic products after 10 days. The mean elimination half-lives found in studies in normal adult subjects (n = 54) were 8.4 hours (range = 3 to 20 hours) for loratadine and 28 hours (range = 8.8 to 92 hours) for the major active metabolite (descarboethoxyloratadine). In nearly all patients, exposure (AUC) to the metabolite is greater than exposure to parent loratadine.

In a study involving twelve healthy geriatric subjects (66 to 78 years old), the AUC and peak plasma levels (Cmax) of both loratadine and descarboethoxyloratadine were significantly higher (approximately 50% increased) than in studies of younger subjects. The mean elimination half-lives for the elderly subjects were 18.2 hours (range = 6.7 to 37 hours) for loratadine and 17.5 hours (range = 11 to 38 hours) for the active metabolite.

Loratadine, dosed once daily, had reached steady-state by the fifth daily dose. The pharmacokinetics of loratadine and descarboethoxyloratadine are dose independent over the dose range of 10 to 40 mg and are not significantly altered by the duration of treatment.

In the clinical efficacy studies, CLARITIN Tablets were administered before meals. In a single-dose study, food increased the AUC of loratadine by approximately 40% and of descarboethoxyloratadine by approximately 15%. The time to peak plasma concentration (Tmax) of loratadine and descarboethoxyloratadine was delayed by 1 hour with a meal. Although these differences would not be expected to be clinically important, CLARITIN Tablets should be administered on an empty stomach.

In patients with chronic renal impairment (Creatinine Clearance ≤ 30 mL/min) both the AUC and peak plasma levels (Cmax) increased on average by approximately 73% for loratadine, and approximately by 120% for descarboethoxyloratadine, compared to individuals with normal renal function. The mean elimination half-lives of loratadine (7.6 hours) and descarboethoxyloratadine (23.9 hours) were not significantly different from that observed in normal subjects. Hemodialysis does not have an effect on the pharmacokinetics of loratadine or its active metabolite (descarboethoxyloratadine) in subjects with chronic renal impairment.

In patients with chronic alcoholic liver disease the AUC and peak plasma levels (Cmax) of loratadine were double while the pharmacokinetic profile of the active metabolite (descarboethoxyloratadine) was not significantly changed from that in normals. The elimination half-lives for loratadine and descarboethoxyloratadine were 24 hours and 37 hours, respectively, and increased with increasing severity of liver disease.

There was considerable variability in the pharmacokinetic data in all studies of CLARITIN Tablets, probably due to the extensive first-pass metabolism. Individual histograms of area under the curve, clearance, and volume of distribution showed a log normal distribution with a 25-fold range in distribution in healthy subjects.

Loratadine is about 97% bound to plasma proteins at the expected concentrations (2.5 to 100 ng/mL) after a therapeutic dose. Loratadine does not affect the plasma protein binding of warfarin and digoxin. The metabolite descarboethoxyloratadine is 73% to 77% bound to plasma proteins (at 0.5 to 100 ng/mL).

Whole body autoradiographic studies in rats and monkeys, radiolabeled tissue distribution studies in mice and rats, and *in vivo* radioligand studies in mice have shown that neither loratadine nor its metabolites readily cross the blood-brain barrier. Radioligand binding studies with guinea pig pulmonary and brain H_1-receptors indicate that there was preferential binding to peripheral versus central nervous system H_1-receptors.

Clinical trials of CLARITIN Tablets involved over 10,700 patients who received either CLARITIN Tablets or another antihistamine and/or placebo in double-blind randomized controlled studies. In placebo-controlled trials, 10 mg once daily of CLARITIN Tablets was superior to placebo and similar to clemastine (1 mg BID) or terfenadine (60 mg BID) in effects on nasal and non-nasal symptoms of allergic rhinitis. In these studies, somnolence occurred less frequently with CLARITIN Tablets than with clemastine and at about the same frequency as terfenadine or placebo. In studies with CLARITIN Tablets at doses 2 to 4 times higher than the recommended dose of 10 mg, a dose-related increase in the incidence of somnolence was observed. Therefore, some patients, particularly those with hepatic or renal impairment and the elderly, may experience somnolence.

In a study in which CLARITIN Tablets were administered at 4 times the clinical dose for 90 days, no clinically significant increase in the QTc was seen on ECGs.

INDICATIONS AND USAGE CLARITIN Tablets are indicated for the relief of nasal and non-nasal symptoms of seasonal allergic rhinitis.

CONTRAINDICATIONS CLARITIN Tablets are contraindicated in patients who are hypersensitive to this medication or to any of its ingredients.

PRECAUTIONS **General:** Patients with liver impairment should be given a lower initial dose (10 mg every other day) because they have reduced clearance of CLARITIN Tablets.

Drug Interactions: The coadministration of a single 20 mg dose of CLARITIN Tablets (double the recommended daily dose) and a 200 mg dose of ketoconazole twice daily to 12 subjects resulted in increased plasma concentrations of loratadine (180% increase in AUC) and its active metabolite, descarboethoxyloratadine (56% increase in AUC). However, no related changes were noted in the QTc on ECGs taken at 2, 6, and 24 hours after the coadministration of loratadine and ketoconazole. Also, there were no significant differences in clinical adverse events between CLARITIN Tablet groups with or without ketoconazole.

Other drugs known to inhibit hepatic metabolism should be coadministered with caution until definitive interaction studies can be completed. The number of subjects who concomitantly received macrolide antibiotics, cimetidine, ranitidine, or theophylline along with CLARITIN Tablets in controlled clinical trials is too small to rule out possible drug-drug interactions. There does not appear to be an increase in adverse events in subjects who received oral contraceptives and CLARITIN Tablets compared to placebo.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: In an 18-month oncogenicity study in mice and a 2-year study in rats, loratadine was administered in the diet at doses up to 40 mg/kg (mice) and 25 mg/kg (rats). In the carcinogenicity studies, pharmacokinetic assessments were carried out to determine animal exposure to the drug. AUC data demonstrated that the exposure of mice given 40 mg/kg of loratadine was 3.6 (loratadine) and 18 (active metabolite) times higher than a human given 10 mg/day. Exposure of rats given 25 mg/kg of loratadine was 28 (loratadine) and 67 (active metabolite) times higher than a human given 10 mg/day. Male mice given 40 mg/kg had a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) than concurrent controls. In rats, a significantly higher incidence of hepatocellular tumors (combined adenomas and carcinomas) was observed in males given 10 mg/kg and males and females given 25 mg/kg. The clinical significance of these findings during long-term use of CLARITIN Tablets is not known.

In mutagenicity studies, there was no evidence of mutagenic potential in reverse (AMES) or forward point mutation (CHO-HGPRT) assays, or in the assay for DNA damage (Rat Primary Hepatocyte Unscheduled DNA Assay) or in two assays for chromosomal aberrations (Human Peripheral Blood Lymphocyte Clastogenesis Assay and the Mouse Bone Marrow Erythrocyte Micronucleus Assay). In the Mouse Lymphoma Assay, a positive finding occurred in the nonactivated but not the activated phase of the study.

Loratadine administration produced hepatic microsomal enzyme induction in the mouse at 40 mg/kg and rat at 25 mg/kg, but not at lower doses.

Decreased fertility in male rats, shown by lower female conception rates, occurred at approximately 64 mg/kg and was reversible with cessation of dosing. Loratadine had no effect on male or female fertility or reproduction in the rat at doses of approximately 24 mg/kg.

Pregnancy Category B: There was no evidence of animal teratogenicity in studies performed in rats and rabbits. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, CLARITIN Tablets should be used during pregnancy only if clearly needed.

Nursing Mothers: Loratadine and its metabolite, descarboethoxyloratadine, pass easily into breast milk and achieve concentrations that are equivalent to plasma levels with an AUC_{milk}/AUC_{plasma} ratio of 1.17 and 0.85 for the parent and active metabolite, respectively. Following a single oral dose of 40 mg, a small amount of loratadine and metabolite was excreted into the breast milk (approximately 0.03% of 40 mg over 48 hours). A decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when CLARITIN Tablets are administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children below the age of 12 years have not been established.

ADVERSE REACTIONS Approximately 90,000 patients received CLARITIN Tablets 10 mg once daily in controlled and uncontrolled studies. Placebo-controlled clinical trials at the recommended dose of 10 mg once a day varied from 2 weeks' to 6 months' duration. The rate of premature withdrawal from these trials was approximately 2% in both the treated and placebo groups.

REPORTED ADVERSE EVENTS WITH AN INCIDENCE OF MORE THAN 2% IN PLACEBO-CONTROLLED ALLERGIC RHINITIS CLINICAL TRIALS

PERCENT OF PATIENTS REPORTING

	LORATADINE 10 mg OD n = 1926	PLACEBO n = 2545	CLEMASTINE 1 mg BID n = 536	TERFENADINE 60 mg BID n = 684
Headache	12	11	8	8
Somnolence	8	6	22	9
Fatigue	4	3	10	2
Dry Mouth	3	2	4	3

Adverse event rates did not appear to differ significantly based on or race, although the number of non-white subjects was relatively

In addition to those adverse events reported above, the following events have been reported in 2% or fewer patients.

Autonomic Nervous System Altered salivation, increased s altered lacrimation, hypoesthesia, impotence, thirst, flushing.

Body As A Whole Conjunctivitis, blurred vision, earache, e tinnitus, asthenia, weight gain, back pain, leg cramps, malaise, ch rigors, fever, aggravated allergy, upper respiratory infection, angio edema.

Cardiovascular System Hypotension, hypertension, palpitatio cope, tachycardia.

Central and Peripheral Nervous System Hyperkinesia, blephar paresthesia, dizziness, migraine, tremor, vertigo, dysphonia.

Gastrointestinal System Abdominal distress, nausea, vomitin lence, gastritis, constipation, diarrhea, altered taste, increased a anorexia, dyspepsia, stomatitis, toothache.

Musculoskeletal System Arthralgia, myalgia.

Psychiatric Anxiety, depression, agitation, insomnia, paroniria, a impaired concentration, confusion, decreased libido, nervousness.

Reproductive System Breast pain, menorrhagia, dysmenorrhea, v

Respiratory System Nasal dryness, epistaxis, pharyngitis, d nasal congestion, coughing, rhinitis, hemoptysis, sinusitis, sneezin chospasm, bronchitis, laryngitis.

Skin and Appendages Dermatitis, dry hair, dry skin, urticaria, ra ritus, photosensitivity reaction, purpura.

Urinary System Urinary discoloration, altered micturition.

In addition, the following spontaneous adverse events have been r rarely during the marketing of loratadine: peripheral edema; abnor patic function, including jaundice, hepatitis, and hepatic necrosis; a seizures; breast enlargement; erythema multiforme; and anaphylax

DRUG ABUSE AND DEPENDENCE There is no information to indic abuse or dependency occurs with CLARITIN Tablets.

OVERDOSAGE Somnolence, tachycardia, and headache have b ported with overdoses greater than 10 mg (40 to 180 mg). In the e overdosage, general symptomatic and supportive measures sho instituted promptly and maintained for as long as necessary.

Treatment of overdosage would reasonably consist of emesis syrup), except in patients with impaired consciousness, followed by ministration of activated charcoal to absorb any remaining drug. If vi is unsuccessful, or contraindicated, gastric lavage should be per with normal saline. Saline cathartics may also be of value for rapid of bowel contents. Loratadine is not eliminated by hemodialysis. I known if loratadine is eliminated by peritoneal dialysis.

Oral LD_{50} values for loratadine were greater than 5000 mg/kg in r mice. Doses as high as 10 times the recommended clinical doses s no effects in rats, mice, and monkeys.

DOSAGE AND ADMINISTRATION Adults and children 12 years of a over: One 10 mg tablet daily on an empty stomach.

In patients with liver failure, 10 mg every other day should be th ing dose.

HOW SUPPLIED CLARITIN Tablets, 10 mg, white to off-white comp tablets; impressed with the product identification number "458" side; and "CLARITIN 10" on the other; high density polyethylene plas tles of 100 (NDC 0085-0458-03). Also available, CLARITIN Unit-of-Us ages of 14 tablets (7 tablets per blister card) (NDC 0085-0458-01) tablets (10 tablets per blister card) (NDC 0085-0458-05); and 10 x 1 Unit Dose-Hospital Pack (NDC 0085-0458-04).

Protect Unit-of-Use packaging and Unit Dose-Hospital Pac excessive moisture. Store between 2° and 30°C (36° and 86°F).

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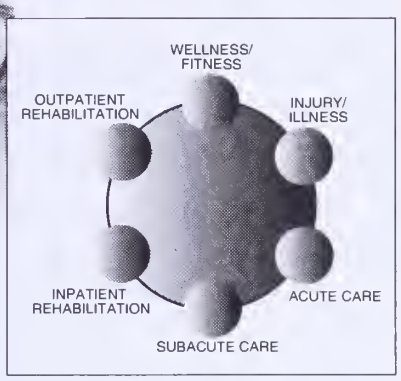

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

National advertising representative: State Medical Journal Advertising Bureau, Inc., 11437 West 106th Street, Overland Park, KS 66214, (913) 888-8781, 1-800-788-2360,

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

MAY 1994 VOL. 87, NO. 5

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Wheat fields ripen in the Oklahoma sun as harvest time approaches.

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EDITORIAL

Ray V. McIntyre, MD

The Parable of the Sorcerer's Apprentice

Nowadays, a wide variety of folks proffer health advice to the ailing patients in our society. From Aunt Mamie and her cranberry juice to the surgeon's coronary rotorooters, there is a vast spectrum of offerings and a consequent wide range of effects. And, in this age of chemistry, an enlarging group of ancillary practitioners are clamoring for the legal authority to prescribe the latest products of the chemistry laboratories. The limited license practitioners now mount frequent legislative initiatives seeking access to the inner sanctum of the nation's pharmacies.Thus the social policy question arises: Who should have the legal authority to prescribe today's potent drugs to human beings? Can a profession-specific formulary provide adequate patient safety?

The ethical answer is that the legal authority to prescribe should be vested in those who have the knowledge and clinical experience to ensure—as far as it is humanly possible—that every prescription will work for the patient's benefit. Medicine is not prescribed for the benefit of the prescriber but solely for the health of the patient. Further, medicines are chemical missiles that do not guide themselves to the eyes, or to the feet, or to the back muscles, but disseminate through the human body according to a complex interaction of anatomy, physiology, and pharmacology. As disease states variably affect human reactions, a broad base of detailed knowledge is necessary for most therapeutic decisions to be correct.

Thus we can say that prescriptive authority should be granted to those with an educational experience that includes the theory of pharmacology and biochemistry, and integrates an expert knowledge of human anatomy and physiology with a wise understanding of the natural history of disease. A knowledge of one body part or of one therapeutic system is not enough foundation to administer medicines that affect the whole patient. After all, it is not the attainment of a degree, but the breadth and the intensity of studies in pathology and pharmacology that bestow the competence to prescribe. The complexities of decision-making in medicine range widely, from common-sense simple to the most complicated and esoteric, and the difficult may look deceptively easy to those having incomplete knowledge.

In considering prescribing authority, there is a parable in the theme of the Disney film short entitled *The Sorcerer's Apprentice*. This well-known musical cartoon, based on a classical song, shows the apprentice magician's terrible misadventures after magically unleashing the natural forces of darkness, lightning, and flood. Unfortunately the student has not yet learned how to direct, control, and contain these unruly forces, and disaster reigns until the master magician returns. The apprentice was at a stage of learning where he... "knew not that he knew not."

Regardless of the degree held and the profession practiced, for the safety and well-being of the patients, prescriptive authority should be vested only in those who have mastered the equivalent of medical school courses in anatomy, physiology, biochemistry, pharmacology, and the natural history of human disease.

Ray V. McIntyre, M.D.

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President's Page

Jay A. Gregory, MD

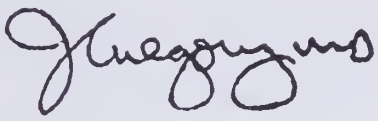
Where Do We Go from Here?

G. Lance Miller, MD, has completed his term as president of the OSMA, and to him we all owe a great deal of gratitude. Without his ability to make tough decisions during these tough times, we would not be where we are today. The inscription on the plaque presented to Dr. Miller at Shangri La read, "The right man, in the right place, at the right time." Dr. Miller, thank you. By your leadership and by the House of Delegates' approval, the Oklahoma physicians network (the IPA) and the managed care organization will become a reality.

Our speaker and vice-speaker of the House of Delegates must be commended for their outstanding job of conducting the business of the House during the Annual Meeting. Those who served on reference committees did an incredible job in putting together their reports for the House after hearing much heated debate on the issues. OSMA staff must be congratulated for putting on another excellent Annual Meeting, although as the meeting went on we became aware of areas where next year's meeting will be even better.

The burden that I feel in writing this letter can not be expressed in words, nor am I so sure that what I feel, if written, would be published. I ask you, what has happened to the "OLD" attitudes of respect, honor, dignity, and courteousness when it comes to interacting with our peers??? The Annual Meeting is a time when we, the elected officials, come together to debate the issues, to establish policy, and to elect new leaders— all of which are to make us stronger, more united in purpose on behalf of our patients and the OSMA membership. Veiled threats, rumors, innuendoes, etc., have no place in the conduct of our business!!! During the meeting I was asked my thoughts on the idea of putting forth a Code of Conduct for the House. My knee-jerk response: good idea. My 48-hour deliberation response: if you as an adult in our society today need a manual to tell you how to interact with your fellow man, need I say more?

Be assured that by the time you read this letter, events will have taken place to resolve some of this conflict. I do not believe that it will be humanly possible to resolve all of this conflict in meetings or by establishing codes of conduct. It is up to each one of us individually to resolve these issues in our own hearts. When that is done, I have faith that you, my friends, will do what is right on behalf of your patients and your fellow physicians. There are no losers in the field of medicine; we are all winners. We have been given the ability to relieve the pain and suffering of our fellow man. Let not our actions create pain and suffering in our fellow physicians. We must now all roll up our sleeves and respectfully resolve the issues that are before us. I eagerly look forward to working with and on behalf of each of you. I thank you for the confidence you have placed in me. I ask that you pray that your God will give me strength and guidance in the months ahead.



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SCIENTIFIC

Urinary Incontinence in Elderly Females

Marie A. Bernard, MD

This article reviews recent literature regarding the incidence, causes, and treatment of urinary incontinence in elderly women. Data gathering was based upon a Medline search of the medical literature from 1987 to 1993. Studies that included fifty subjects or more and that were published in a physician-oriented journal were selected for inclusion. The findings indicate that urinary incontinence is a commmon problem among elderly women and one that can be easily evaluated and treated, at least initially, in any physician's office.

URINARY INCONTINENCE IS A FETID, DIRTY, EMBARrassing issue that often causes elderly women to lead a restricted social life.[1-3] It is a problem that one associates with the aging process. However, urinary incontinence is not a necessary accompaniment of aging.[4-7] It is a problem for which there are some easily implemented interventions.

The goal of this review is to discuss results of recent studies evaluating the incidence, causes, and treatment for urinary incontinence in elderly women.[1] Emphasis will be placed on the recently disseminated guidelines for the management of urinary incontinence, developed by the Agency for Health Care Policy and Research (AHCPR).[8]

Prevalence of the Problem

What is the prevalence of urinary incontinence, particularly among the elderly? Bauer examined this question in Denmark in a mailed questionnaire survey of 600 women ages 20 to 79 years.[9] Sixty-nine percent of eligible women responded to the questionnaire. The overall incidence of incontinence in this population was 34%, with the frequency ranging from 4.8% in women in their thirties, to 9.2% in women in their sixties. Diokno et al surveyed noninstitutionalized elderly (greater than 60 years old) in Washtenaw County, Michigan.[10] Of 3,005 eligible seniors, 65% participated in the household interview (N=1,956). They documented an incidence of incontinence in females that varied between 35 and 41%. When octogenarians are surveyed, similar numbers are obtained.[11]

Ouslander and colleagues evaluated the incidence of incontinence in community-dwelling dementia patients.[12] They surveyed 184 caregivers of demented individuals. Thirty-six percent of the patients had urinary incontinence. Incontinence was more likely in demented individuals with fewer years of formal education (11.1 years versus 12.7 years, $P<0.05$), and in those with a lower mini-mental status exam score (11.42 versus 15.57, $P<.001$; normal = 24 or higher). As might be anticipated, the perceived burden of care, and the perceived likelihood of institutionalization, was greater among the caregivers of incontinent patients than among the caregivers of continent patients. In follow-up, 39% of the patients had been institutionalized. Incontinence was found to be a significant contributing factor in the decision to institutionalize, although it was not the primary factor.

In short, urinary incontinence is a prevalent prob-

Direct correspondence to Marie A. Bernard, MD, University of Oklahoma College of Medicine, PO Box 26901, 4SP-120, Oklahoma City, OK 73190.

lem in the elderly population. It increases in frequency with advancing age, in the demented, and in institutionalized populations.

Etiology of Urinary Incontinence

What causes urinary incontinence in the elderly? As very nicely discussed by Resnick,[13] the control of micturition is complex, and dependent upon an intact nervous system, detrusor muscle, and urethral sphincter. The entire nervous system plays a role in the control of micturition, including the cerebral hemispheres, the spinal cord, and the local nervous system. Disruption at any of these levels could lead to difficulties with voiding. Common disorders among the elderly which may contribute to such disruption include stroke, diabetic autonomic neuropathy, and tumor of the cerebral hemispheres or spinal cord. In addition, the detrusor muscle, the major muscle lining the bladder, is pivotal for the control of continence, just as the diaphragm is essential for the control of respiratory function. Disorders such as diabetic autonomic neuropathy, overdistension of the bladder due to obstruction, or pharmacologic agents used to treat systemic disorders can contribute to detrusor muscle dysfunction. The urethral sphincter is the third major component in the control of micturition. Loss of competence of the sphincter can occur due to prior trauma or surgery. In females, it has been demonstrated that decreases in systemic estrogen levels can predispose to problems with urinary sphincter competence. Additionally, pelvic muscle relaxation, as a result of multiple pregnancies, can lead to significant problems with maintaining continence, as a result of bladder prolapse.

Levy and Wight have demonstrated that there are structural changes within the submucosa of the bladder that may have implications for maintaining continence.[14] They examined bladder samples from 35 human bladders, aged 6 weeks to 95 years. They found that with age, collagen fibers of the submucosa separate from one another and no longer form fascicles. These changes are similar to those found in other tissues, where the collagen disruption leads to decreased tissue compliance. With aging, there is the accumulation of electron dense material and cellular debris. These changes are also accelerated by obstruction. Finally, they found that smooth muscle cells of the muscularis mucosa layer develop vesicles reminiscent of signs of cell death seen in other tissues. Whether or not these changes contribute to the development of urinary incontinence is not yet clear. However future research may help to illuminate this.

A number of studies show a correlation between medical illnesses and urinary incontinence in the elderly. Diokno[10] examined the historical relationship between medical problems and the occurrence of incontinence. They found that transient ischemic attacks and paresthesias were significantly correlated ($P<0.05$) with the occurrence of urinary incontinence. Hellstrom[11] examined the historically reported incidence of similar illnesses and found confirmatory data. Neurologic disease, cardiovascular disease, respiratory disease, and urinary tract or genital illnesses all strongly correlated with the likelihood of urinary incontinence.

Evaluation of Urinary Incontinence

How should an elderly female with incontinence be evaluated? The physician should be aware that the patient may not feel comfortable discussing problems with continence and may not voluntarily raise the issue.[1-3] Thus, routinely questioning elderly women regarding continence, using open ended questions such as "Do you have trouble with your bladder?" and "Do you have trouble holding your urine (water)?" may be desirable.[8] A thorough history should be obtained from women with incontinence; it should include probing for: past history of neurologic or genital problems; history of medications, both prescription and over-the-counter; and the exact settings in which the incontinence occurs.

An incontinence chart to evaluate the frequency of incontinence episodes and circumstances surrounding the episodes can be an invaluable aid in the evaluation of incontinence. These charts can be utilized by a cognitively intact patient, as well as by the caregiver of a patient who is cognitively impaired. A complete physical examination should be performed, including abdominal examination, with estimation of post-void residual; pelvic examination for atrophy, prolapse, tenderness, muscle tone, hypermobility (posterior rotational descent of the proximal urethra and bladder neck when the patient strains or coughs); and rectal examination, evaluating rectal tone, masses, and evidence of fecal impaction. In addition, there should be laboratory studies to determine the likelihood of reversible metabolic abnormalities or infections that may have predisposed the patient to problems with incontinence. A post-void residual should be assessed, to determine whether the patient suffers from significant obstruction. A post-void residual greater than 200 ml suggests the need for immediate evaluation of obstruction.[8]

Urinary tract infection must be ruled out as a cause

of incontinence. However, the presence of infection is sometimes difficult to determine. Twenty percent of women ages 65 to 70 years have asymptomatic bacteriuria. Twenty-three to 50% of women more than 80 years old have this problem.

It has been demonstrated that asymptomatic bacteriuria is a dynamic problem in the elderly. Boscia et al studied 260 individuals more than 67 years old[15] with three surveys for bacteriuria, six months apart. They demonstrated that 16% of women had bacteriuria at baseline. Thirty percent of women had bacteriuria at one of the three surveys. However, only 6% of women had persistent bacteriuria. Given this dynamic aspect of bacteriuria, it is difficult to determine whether bacteriuria at any one time may be leading to problems with urinary incontinence.

Additionally, Boscia demonstrated that elderly individuals may have symptoms (with the exception of dysuria) suggestive of urinary tract infection, whether or not bacteria are present. The frequency of problems with urgency, frequency, and nocturia were the same for individuals with and without bacteriuria. Consequently, in the absence of pyuria or dysuria, it is probably not necessary to treat bacteriuria in the elderly. It may, in fact, be harmful, given the small therapeutic window for medications in the elderly. However, whenever there is a question of a possible urinary tract infection, it is common practice to treat with one course of antibiotics, and then assess the response. If there is no response, further attempts at obliterating bacteria within the urine are probably not beneficial.

After the initial evaluation, and the exclusion of temporary and reversible causes of the incontinence, it is worthwhile attempting to classify the type of incontinence. Incontinence can be classified as follows[8]:

1. Stress incontinence—involuntary loss of urine during coughing, sneezing, laughing, or other physical activity; confirmed by observing loss during activities that cause increased abdominal pressure (stress);

2. Urge incontinence—involuntary loss of urine associated with an abrupt and strong desire to void (urgency); usually associated with urinary frequency;

3. Overflow incontinence—involuntary loss of urine associated with overdistension of the bladder (overflow), and associated with post-void residuals of greater than 200 ml; this form of incontinence uniformly requires either surgical relief of obstruction, or various catheterization methods to assure complete emptying of the bladder;

4. Mixed incontinence—combination of the three major categories of incontinence, often found in frail elders.

The Agency for Health Care Policy and Research recently convened an expert panel to develop guidelines for the evaluation and management of individuals with urinary incontinence.[8,16] The consensus of these experts for the approach to stress and urge incontinence in females is summarized in Figures 1 and 2. For women with a mixed disturbance, the approach should be in keeping with the predominant symptoms.

The Role of Urodynamics.—Urodynamics are tests which are performed to determine the anatomic and functional status of the bladder and urethra. These tests include uroflowmetry, which measures the urine flow rate visually, electronically, or with the use of a disposable unit; and cystometry, which measures bladder sensation, capacity, and compliance, and can be used to assess voluntary and involuntary detrusor contractions. These tests can be performed simply at the bedside or in a urodynamics laboratory with extensive electronic monitoring. The more elaborate forms of this testing generally require a cognitively intact subject who is able to assume a prone position.

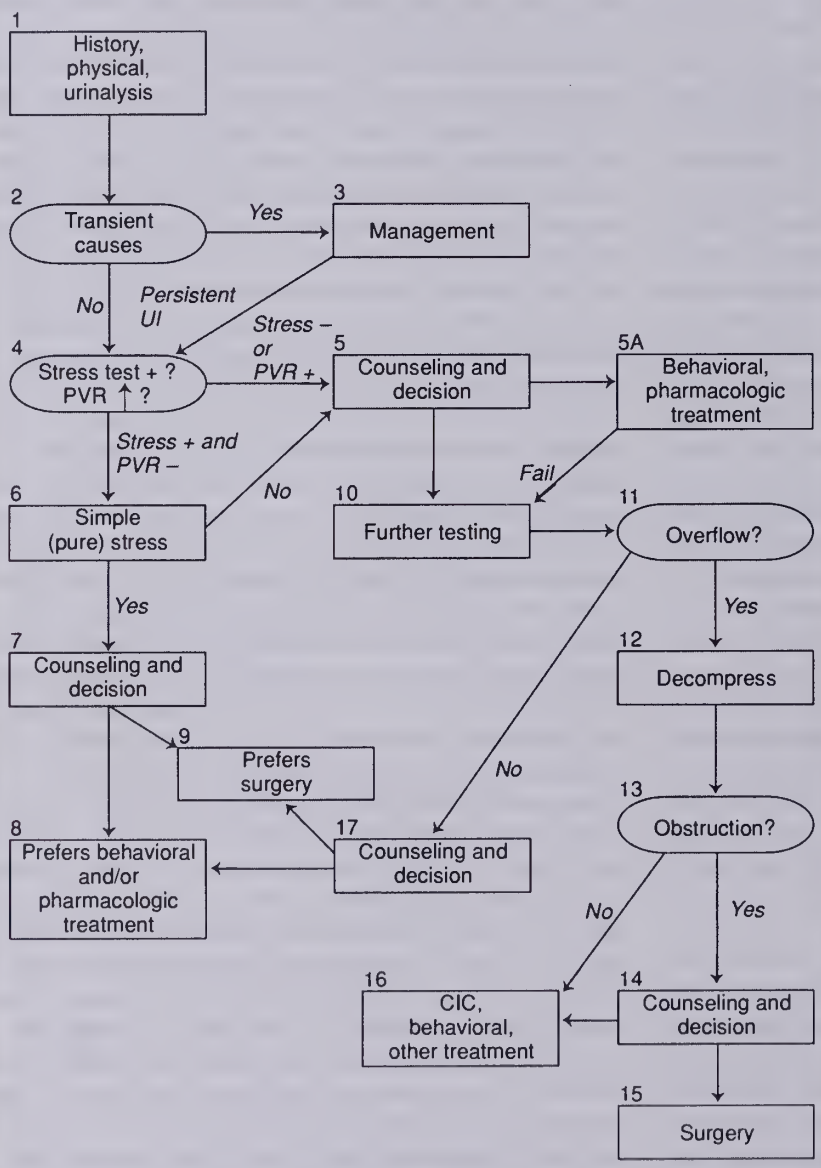
What is the role of urodynamic evaluation in the elderly? Fantl et al evaluated a group of 145 incontinent women at Medical College of Virginia.[17] These women were volunteers obtained from newspaper advertisements for behavior modification of urinary incontinence. They were 55 years of age and older, cognitively intact, with urinary incontinence for at least one week. Fifty-seven percent had undergraduate college degrees or more, and 58% had an annual income of $20,000 or more. The women underwent an evaluation that included a pelvic exam, retrograde cystometrogram, urethral function evaluation, and stress testing for stress incontinence.

In spite of the highly educated subject group, urodynamic evaluation showed no evidence of a diagnosable urologic abnormality in 10% of the population. Of urodynamic diagnoses that were established, 62% of the population (N=90) had evidence of stress incontinence, 12% (N=17) had detrusor instability (urge incontinence), and 16% (N=23) had a mixed disturbance. The only significant difference between the groups was that those who had no diagnosable urodynamic abnormality were younger (67±7 years old) than those individuals with mixed disturbances (73±9 years old, $P<0.05$). They further evaluated the subjects to correlate symptomatology to the urodynamic diagnosis, and found no reliable relationship between symptoms and urodynamics. However,

the severity of incontinence did correlate with the urodynamic diagnosis, with the most severe incontinence occurring in women with mixed disturbances and the least severe in women with no urodynamically demonstrable abnormality ($P<0.01$).

Yu et al did a similar evaluation of 133 female nursing home residents.[18] Nursing assistants had documented the baseline incidence of incontinence via hourly checks, 14 hours per day, 7 days per week, for 3 weeks. Nonetheless, when urodynamic evaluation was done, 41% of patients had normal cystometrograms. The incidence of incontinence was found to correlate strongly to mobility status, with 68% using a wheelchair, 18% using a walker, and 61% requiring a geri chair or restraining device. (Some patients required more than one modality, thus the numbers do not add up to 100%.) Urinary incontinence in this population also strongly correlated with mini-mental status exam score, with 47% of participants having mini-mental status exam scores of 0-10 (consistent with severe cognitive impairment) and 22% having mini-mental status exam scores of 11-17 (moderate cognitive impairment).

Figure 1. Female Stress Incontinence



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Dennis et al evaluated 155 females in nursing homes in Gloucester, New Jersey.[19] They found a similar relationship between mini-mental status exam scores and the incidence of incontinence. They also found that activity level strongly correlated with incontinence, but did not correlate with cystometrogram findings. Almost half of the individuals who were totally dependent for all activities of daily living had normal cystometrograms. Interestingly, at follow-up urodynamic evaluation 13 weeks later, without any therapeutic intervention, 45 of the 155 patients (33%) had urodynamic findings which differed from their initial evaluation. Almost half of these now had normal cystometrograms, while the others had changed from a normal baseline evaluation to an abnormal evaluation.

Diokno et al evaluated the sensitivity and specificity of urodynamic evaluation.[20] They recruited 167 individuals, 60 to 86 years old, to undergo urodynamic testing including post void residual, cystometry, passive and dynamic urethral profilometry (both supine

and standing), lateral cystometrograms (sitting and standing), and measurement of the anatomic urethral length. They found no significant difference in urodynamics of continent and incontinent participants, except for the stress test. They found the stress test was 98% specific, but only 40% sensitive in this patient population.

Thus one must question the benefit of urodynamics, beyond a stress test, early in the evaluation of elderly incontinent patients. Urodynamic testing is thought to be necessary prior to referral of a patient for surgery, and in the presence of marked pelvic prolapse, severe stress incontinence, significantly elevated post-void residuals, diabetes mellitus, and in the presence of microhematuria.[21-23] Please refer to Figures 1 and 2 for the AHCPR recommended role of urodynamics in the process of evaluation of the elderly female with incontinence.

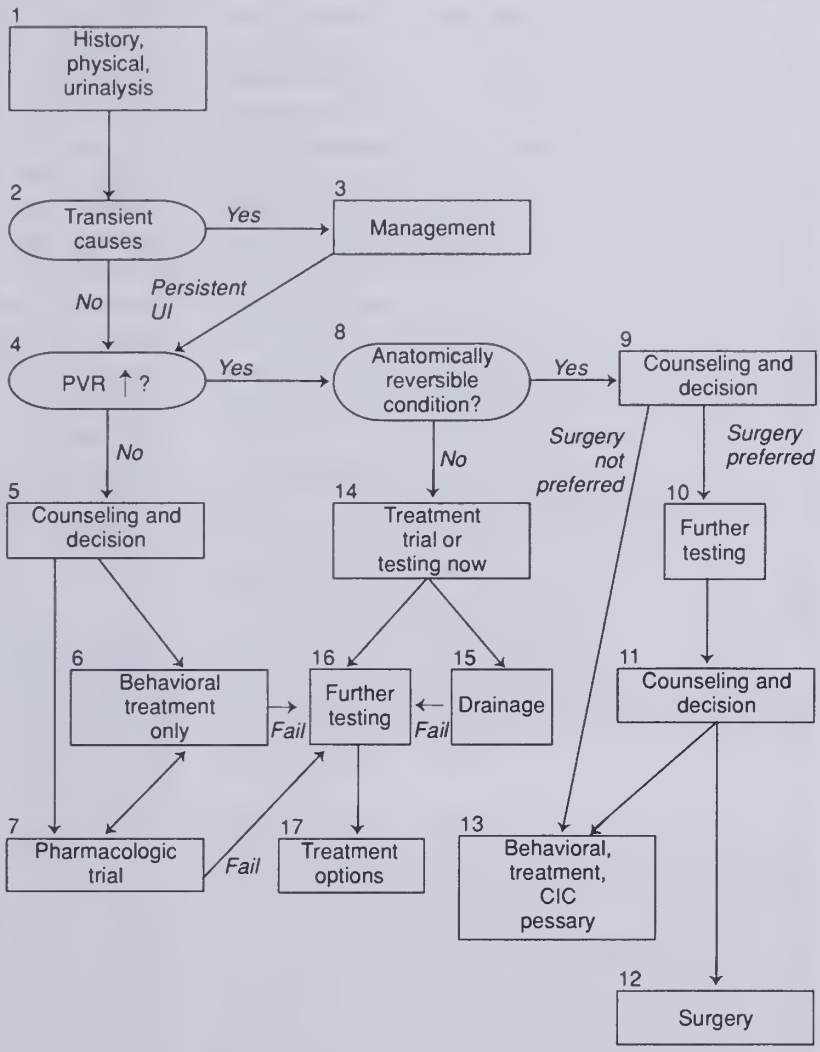
Treatment of Urinary Incontinence

What should be the appropriate treatment of elderly females with urinary incontinence? As noted above, every individual deserves a thorough history and physical, including a search for evidence of urinary retention, fecal impaction,[24] or severe cystocele or rectocele. However, in the absence of obviously reversible factors, the recommended approach is to consider behavioral intervention, pharmacologic therapy, or surgery.

Behavioral Intervention.— Non-pharmacologic therapy for urinary incontinence in elderly females should probably be the first consideration, regardless of the etiology of the incontinence, unless the patient suffers from overflow incontinence, severe cystocele or rectocele, or is otherwise a candidate for immediate surgery.[25-28] Such intervention may consist of pelvic muscle exercises for strengthening (Kegel exercises) or bladder training by timed voidings.

Pelvic muscle (Kegel) exercises improve urethral resistance through active exercise of the pubococcygeus muscle. The patient is taught to "draw in" the perivaginal muscles and anal sphincter as if to control urination or defecation, but without contracting abdominal, buttock, or inner thigh muscles. Emphasis is placed on sustaining contractions for up to 10 seconds, followed by an equal period of relaxation, for up to 30 to 80 repetitions per day. This is carried on intensively for at least six weeks, and often longer for elderly women. Success with this in-

Figure 2. Female Urge Incontinence



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tervention is greatest when a trained health educator supervises the intervention.[15,29]

Bladder training consists of educating the patient regarding the physiology of micturition and then establishing a program of scheduled voiding, with positive reinforcement for success at delaying the urge to void. This form of intervention has been found to be successful for both urge and stress incontinence.[30,31] As is implicit, the greatest success with this intervention is associated with a trained health educator supervising the intervention.

McClish evaluated the urodynamics of elderly individuals who underwent bladder training exercises.[32] Their program was one of scheduled mandatory micturition with avoidance of voluntary bladder emptying between voiding intervals. The study participants initially voided every 30 to 60 minutes. They ignored the urge to void between scheduled voidings with the use of relaxation techniques and distraction. The interval between voidings was increased by 30 minutes weekly, aiming at 2.5- to 3-hour intervals. The intervention lasted for six weeks, with weekly clinic visits for compliance review, encouragement, and positive reinforcement. In the 108 female subjects, there was significant improvement in the first sensation to void, and in the maximal cystometric capacity versus the first sensation to void ($P<0.001$). More important, the number of incontinent episodes per week and the amount of fluid lost with each episode of incontinence were significantly decreased by the end of treatment ($P<0.001$).

A similar study by Baigis-Smith involved 54 volunteers.[33] Study participants underwent nurse practitioner-supervised Kegel exercise instruction, with the use of a perineometer for feedback. They were seen every 2 weeks until there was improvement demonstrated in the force of perineal muscle contractions. The patients were then reevaluated at 1 month, 3 months, 6 months, and 1 year. At each follow-up visit the incontinence record for the previous 2 weeks was reviewed, and the subject received biofeedback by the perineometer. The incidence of accidents per week was improved from 17.4 on average at baseline to 4.2 at the end of treatment. At the 1-year follow-up, the incidence of accidents per week was only 1 per week. However, there was significant dropout among participants, from 54 at the end of the active intervention to 19 at the 1-year follow-up visit.

Hu et al evaluated the impact of behavior therapy for urinary incontinence in 134 female nursing home patients.[34] The intervention involved a nursing assistant checking the patient every hour, fourteen hours per day, and reminding the patient to ask to go to the bathroom. When the subject successfully waited to void for one hour, there was positive reinforcement with praise, time spent conversing with the patient, or providing additional services. However, the patients were encouraged to call for help to go to the bathroom if it was needed before the next hour's check. There was a significant decrease in the incidence of incontinence in study participants versus controls. The mean number of incontinent episodes per day was decreased by 0.6 in the experimental group. The best results were obtained in those individuals who had normal cystometrograms, who had higher cognitive function, and who had the highest frequency of incontinence at baseline. Hu notes that a great deal of the documented improvement could be attributed to the clients' learning to ask for help to get to the bathroom. Subsequent studies have documented that attitudes of nursing home staff and administration also play a significant role in the success of an intervention such as this.[35]

The expert panel involved in establishing the AHCPR guidelines for management of urinary incontinence found that 87% of women were cured or improved with pelvic muscle exercise, while 70% were cured/improved with bladder training.[16] As expected, there were no side effects or complications associated with either form of therapy.

Other interventions, such as electrical stimulation of pelvic muscles to increase pelvic and bladder muscle tone, have been attempted.[36] However, such interventions are not considered first line therapy for elderly females.

Drug Therapy.—There are a number of drugs available for the treatment of urinary incontinence.[37] AHCPR recommended drugs are listed in Table 1.[8]

Drugs that act on the urethra help with stress incontinence. Alpha-adrenergic agents can stimulate alpha-receptors in the bladder neck and urethra and

Table 1. Drugs for Treatment of Urinary Incontinence

Stress Incontinence
Alpha-Agonists
• Phenylpropanolamine 25–75 mg, p.o., q12 h
Urge Incontinence
Anticholinergics
• Propantheline 7.5–30 mg in the fasting state, t.i.d.–q.i.d.
• Qxybutinin (FDA approved) 2.5–5 mg, p.o., t.i.d.
• Imipramine (FDA approved for enuresis in children) 10–100 mg p.o./d, initially in divided doses
• Doxepin 10–100 mg p.o./d, initially in divided doses
• Dicyclomine hydrochloride 10–20 mg, t.i.d., p.o.

theoretically may be beneficial for urinary incontinence. Alpha-adrenergic agonist agents should be used with caution in patients with hypertension, hyperthyroidism, cardiac arrhythmias, and angina. Alpha-agonists have been found to cure or improve stress incontinence in 19 to 74% of women, based upon review of the literature.[16] Side effects occurred in 0 to 20% and complications in 5 to 33%.

Anticholinergic drugs are commonly used for urge incontinence. They effect the micturition reflex with competitive blockade of muscarinic receptors at postganglionic parasympathetic receptor sites. It should be noted that all anticholinergic drugs are contraindicated in patients with narrow-angle but not wide-angle glaucoma. Anticholinergics have been reported to cure or improve urge incontinence in 0 to 83% of women, with 0 to 70% experiencing side effects.

Tricyclic antidepressants, in particular, imipramine, have both an anticholinergic and direct muscle relaxant effect which may be beneficial.

There are estrogen receptors in the lower urinary tract, bladder, trigone, and distal urethra. Both local and systemic estrogens have been demonstrated to improve urethral pressures, and the proliferation of urethral mucosa. Estrogen replacement is recommended cyclically with conjugated estrogen, 0.3 to 1.25 mg/d, P.O., or vaginally, 2 g or fraction/d. Progestin should be provided with the estrogen if endometrial tissue is present, in doses of 2.5 mg/d continuously, or 10 mg/d intermittently. Recent data suggest that intravaginal estrogen therapy may also prevent recurrent urinary tract infections in postmenopausal women.[38]

Table 2. Surgical Management of UI

UI Type	Cause	Treatment
Stress	Hypermobility	Retropubic suspension Needle endoscopic suspension
Stress	Intrinsic sphincter deficiency	Sling (mostly female) Artificial sphincter Urethral bulking
Urge	Refractory detrusor instability	Augmentation cystoplasty
Overflow	Obstruction	Relieve obstruction
Overflow	Nonobstructive	Intermittent catheterization Other

Urinary Incontience in Adults: Quick Reference Guide for Clinicians. AHCPR Pub. No. 92-0041. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services, March 1992.

Surgery.—The surgical options for treatment of female urinary incontinence are dependent upon the cause of the problem. Whenever considering surgery as an option, there must be objective documentation of the cause of the incontinence, as well as of the patient's risk for surgery, versus the potential for improvement in quality of life. The options for surgical therapy are noted in Table 2.[7]

Summary

In sum, it can be stated that urinary incontinence is a prevalent problem among elderly women. It is associated with embarrassment and high social costs. It is a problem that can be easily evaluated, at least initially, in any physician's office. This evaluation should include a good history and physical, and an incontinence chart. The role of early urodynamic evaluation in the elderly population should be questioned, given recent data. The role of medications needs to be weighed versus the risks, in view of the high prevalence of chronic conditions among the elderly that may contraindicate many of the drugs which have potential beneficial effects for urinary incontinence. However, behavioral therapy, particularly when sustained, may have a role in the initial treatment of such patients. Ultimately none of these interventions can take the place of surgery in those individuals who require it and are good candidates.

Acknowledgments

The author gratefully acknowledges the assistance of M.D. Andrews, MD; Kami Barton, MD; and W. Parry, MD for review and editorial comment upon this manuscript. Mrs. Kim Lewis is acknowledged for her secretarial assistance.

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The Author

Marie A. Bernard, MD, is associate professor of medicine at the University of Oklahoma College of Medicine and director of the Oklahoma Geriatric Education Center.

Abdominal Pain Secondary to a Lymphangiomatous Cyst of the Adrenal: Case Report and Review of Literature

Marc S. Milsten, MD; John A. Minielly, MD; Patrick VanSchoyck, MD; John B. Forrest, MD

An adrenal cyst is a rare clinical finding with an incidence of approximately 0.06% in the general population. As the majority are clinically silent, adrenal cysts are usually discovered incidentally at autopsy or during surgery for an unrelated disease process. We report a case of a lymphangiomatous adrenal cyst diagnosed in a 27-year-old female presenting with abdominal pain.

Case Report

A previously healthy 27-year-old Caucasian female presented to her family physician with a two-week history of vague abdominal pain. Radiographic studies of the upper gastrointestinal system were normal. A sonogram of the abdomen identified a 7.5 cm diameter spherical, cystic mass in the left upper quadrant located between the tail of the pancreas, the medial margin of the spleen, and the upper pole of the left kidney (Fig. 1). Computerized tomography (CT) of the abdomen demonstrated a 9 cm cystic mass in the posterior aspect of the left upper quadrant with inferior displacement of the left kidney (Fig. 2). CT-directed aspiration of the cyst yielded 300 cc of clear, yellow fluid. Postdrainage CT imaging demonstrated replacement of the cystic mass by the tail of the pancreas, suggesting the possibility of a pancreatic pseudocyst. Results of fluid analysis, however, were not consistent with a pseudocyst. Bacterial cultures and cytology were within normal limits. Four weeks

Figure 1. Sonogram demonstrating upper pole cystic mass.

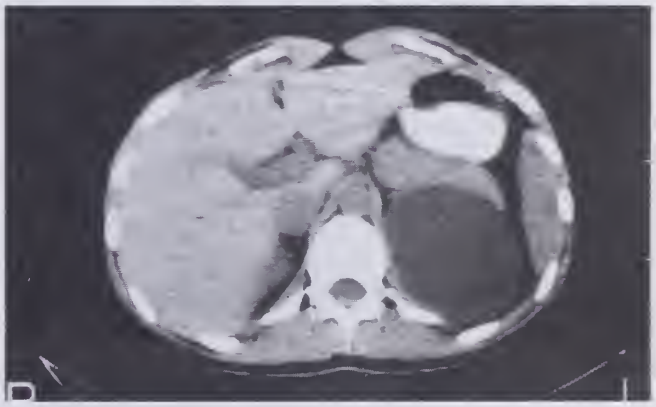

Figure 2. Abdominal CT scan demonstrating cystic lesion at superior pole of left kidney.

From the Department of Surgery, Division of Urology, University of Oklahoma Tulsa Medical College, and St. John Medical Center, Tulsa, Oklahoma.

Direct correspondence to John B. Forrest, MD, St. John Physicians Building, Suite 801, 1725 East 19th, Tulsa, OK 74104.

later, because of recurrent abdominal pain, a repeat sonogram revealed recurrence of the cyst with enlargement to 8 cm in diameter. Intravenous pyelography, cystoscopy, and retrograde pyelography were all within normal limits. Prominent thickening of the wall of the cyst and multiple septa were noted. Because of the rapid reaccumulation of fluid and increasing abdominal symptomatology associated with the mass, the patient was admitted for surgical exploration of the left flank.

The left kidney and adrenal gland were explored through a flank incision. This revealed a partially decompressed, cystic lesion arising from the posterior lateral margin of the left adrenal gland. The cyst and a small portion of the adrenal tissue were removed en bloc. Frozen section examination of the specimen revealed no evidence of malignancy. The patient tolerated the procedure well without complications. She was discharged from the hospital on the fifth postoperative day. Two months postoperative, the patient was asymptomatic and had resumed normal activities.

Pathology

The resected adrenal mass weighed 40 grams and measured 6 cm in maximal dimension. The adrenal gland was almost completely replaced by a smooth-walled cyst that contained clotted blood (Fig. 3). Microscopically, the wall of the cyst consisted of mildly chronically inflamed fibrous tissue with hemosiderin pigmentation and a few remnants of adrenal cortical and medullary tissue. Focally, on the internal surface, there were a few flattened cells, suggestive of endothelial type (Fig. 4). There was no evidence of any preexisting neoplasm.

Since the aspirated fluid was colorless, it is probable that the intracystic hemorrhage occurred secondary to the aspiration, and the cyst is consistent with a lymphangiomatous type.

Discussion

Adrenal cysts rarely present clinically in adulthood. The first reported case of a symptomatic adrenal cyst was in 1670 by Greisellus, who reported a ruptured adrenal as the cause of death of a 45-year-old man.[1] Wahl cited nine cases of adrenal cysts among 14,000 autopsies for an incidence of 0.06%.[2] As of 1977, only 280 cases of adrenal cysts in adults had been reported. The majority are discovered incidentally at autopsy or at surgery for unrelated disease. Adrenal cysts are equally distributed from right to left and are bilateral in 8% of cases. They occur more frequently between the third and fifth decades with a two- to three-fold predominance in females.

The majority of adrenal cysts are clinically silent because, in part, their size rarely exceeds 10 cm in diameter.[4] When significant enlargement of a cyst occurs, symptoms may result from compression of surrounding structures. Abeshouse and colleagues described a triad of findings that included flank discomfort, a palpable mass in the homolateral flank, and downward displacement of the corresponding kidney on x-ray.[5] A more dramatic presentation of an acute abdomen can also emerge with hemorrhage into a cyst or traumatic rupture of its walls.[6] Differential diagnosis includes anomalies of the kidneys, such as renal cysts, carcinoma, hydronephrosis of an upper renal segment, and, rarely, a pedunculated hypernephroma of the upper pole of the kidney.[7] Consideration also includes tumors and cysts of the pancreas, spleen, liver, and mesentery.

Radiographic studies are central to the evaluation of a possible adrenal cyst. The initial approach to di-

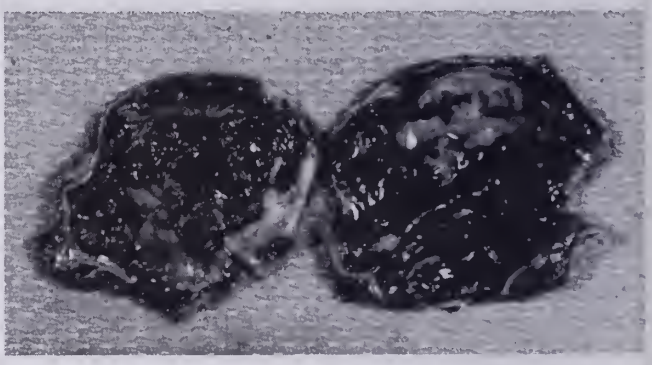

Figure 3. Resected adrenal gland, most of which has been replaced by hemorrhagic cyst.

Figure 4. Internal lining of cyst containing fibrous tissue covered by a thin layer of flattened endothelial-like cells (H&E stained section, original magnification 320×).

agnosis includes plain abdominal x-rays and intravenous pyelography. Calcification occurs microscopically in 60% of cysts, and may be observed radiologically in up to 10% of plain films.[6] Biochemical studies may be necessary to rule out a functional adrenal tumor. Other specialized diagnostic techniques include nephrotomography, renal and adrenal sonography, selective arteriography, and computerized tomography with CT-directed biopsy or aspiration.[3]

Cysts of the adrenal glands are categorized pathologically according to a classification proposed by Foster in 1966. He summarized the nomenclature and incidence of the lesions as follows: parasitic (7%), epithelial (9%), endothelial (45%), and pseudocysts (39%).[8] Endothelial cysts are divided into lymphangiomatous cysts and angiomatous cysts. The rare angiomatous type arises from dilated capillaries or sinusoids between columns of adrenal cells.[3] Lymphangiomatous cysts are characterized by a smooth, flattened endothelial lining. They occur secondary to faulty development of lymphatic, lymphangiectasis of minute lymph spaces, or cystic degeneration of a hamartoma. Lymphatic cysts may be multiple and small, ranging from 1 to 15 mm in diameter or can attain much larger dimensions, as was our experience.[3]

While the endothelial cysts comprise the statistical majority, pseudocysts are the most common type of adrenal cysts recognized clinically. They develop, following hemorrhage, into a normal adrenal gland or extravasation into a pathologic gland or tumor such as a pheochromocytoma.[6,7]

Treatment involves removal only for relief of symptoms, control of associated hypertension, or if rupture is suspected. Percutaneous aspiration of the cystic contents allows cytologic analysis of the fluid and can provide relief of symptoms. In our case, CT-directed drainage resulted in temporary relief of symptoms for the patient but was followed by a prompt recurrence of the cyst. Surgery may be necessary, therefore, in the investigation and treatment of an adrenal cyst.

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The Authors

Marc S. Milsten, MD, was a medical student with the University of Oklahoma Honor Service when this paper was written. He is now chief resident at the University of Kansas, Department of Urology.

John A. Minielly, MD, is a specialist in anatomic and clinical pathology in Tulsa.

Patrick VanSchoyck, MD, Tulsa, has a private practice in family medicine.

John B. Forrest, MD, is a urological surgeon in Tulsa.

Rheumatoid Arthritis: A Complication of Interferon Therapy

Farah Nadir, MD; Stefano Fagiuoli, MD; Harlan I. Wright, MD; Abdul Nadir, MD; Evelyn Hopp, RN; Judith Gavaler, PhD; David H. Van Thiel, MD

A case report of rheumatoid arthritis developing during α-interferon therapy of chronic hepatitis C is reported.

The rheumatoid arthritis was severe, being non-responsive to the use of nonsteroidal anti-inflammatory drugs, and persisted despite discontinuation of the interferon therapy. The literature relative to this case suggesting an association between rheumatoid arthritis and interferon therapy is presented. This case suggests that interferon therapy may precipitate subclinical rheumatoid arthritis in an individual without pre-existing clinical arthritis.

INTERFERONS ARE A GROUP OF GLYCOPROTEINS POSsessing antiviral, immunomodulatory, and antiproliferative activities.[1] Clinically, they have been shown to be useful in the treatment of certain viral conditions, some autoimmune diseases, and various cancers.[2-7]

Occasionally a putative autoimmune disorder is precipitated or induced as a consequence of α-interferon (IFN) treatment.[8-16] Thus, Ronnblom et al reported an incidence of 19% of new autoimmune diseases occurring in patients with malignant carcinoid who were treated with IFN.[17] A similar incidence of new rheumatic diseases was reported by Wandl et al in patients with chronic myelogenous leukemia and essential thrombocythemia who were treated with IFN.[18] In both of these reports and in several others, autoimmune thyroid disease appears to be the most frequently induced disease process.[8,9,15,17,18] Systemic lupus erythematosus, pernicious anemia, psoriasis, sarcoidosis, autoimmune hemolytic anemia, and vasculitis are other diseases that have been induced by IFN.[10-12,16,17]

Despite the fact that rheumatoid arthritis (RA) is a very common autoimmune disorder, few cases of RA have been reported in patients receiving IFN therapy. Reported here is a case of new onset severe RA occurring in a young adult woman free of any prior history of autoimmune disease, who received IFN for the treatment of hepatitis C virus (HCV) positive chronic active hepatitis.

Case Report

A 39-year-old woman with a history of chronic persistent hepatitis, presumably acquired as a consequence of one or more needle sticks while working in a hospital, was first seen in 1978. Her hepatitis serologies (HBV) were negative. For several years, she experienced on and off symptoms of fatigue, nausea, and right upper quadrant pain. Her liver function tests varied from normal to four-fold elevated. In 1992, she was tested for hepatitis C antibody using a second generation ELISA system and was found to be positive. At the same time, she tested negative for rheumatoid factor, antinuclear antibody, antimitochondrial antibody, and antismooth muscle antibody. Her HLA genotype was HLA A3, 33, B7, 14 and DR1, 10. She was started on IFN at a dose of 5 million units 3 times a week. During the sixth month of therapy, she developed severe symmetrical polyarthralgias of

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Oklahoma.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112.

her distal and proximal interphalangeal points, elbows, and knees. This progressed to frank arthritis. She was retested and found to be positive for rheumatoid factor, to have an ESR of 101, and a newly positive ANA with a titer of 1:100 (speckled pattern). C3 and C4 levels were normal (84 and 20, respectively). A diagnosis of rheumatoid arthritis was made. The initiation of therapy for RA with nonsteroidal anti-inflammatory drugs (NSAIDs) and low-dose prednisone (5 mg every day) did little for her symptoms. Only after her IFN therapy was discontinued did she began to experience some relief of her symptoms of RA. Ten months after stopping all IFN, she continues to be a positive RF and have severe symptoms of RA.

Discussion

The central event in the development of autoimmune disease, whatever the specific target organ might be, appears to be the proliferation of high affinity self-reactive T cells. The specific autoimmune disease produced is the consequence of one or usually several different effector mechanisms that recognize the specific organ involved as a target. Specifically, certain lymphokines such as TNFα can injure organ-specific cells directly while others, like IFN, activate T cells and macrophages, which are responsible for tissue damage. Moreover, self-reactive T cells are a continuing source of interleukin 2 (IL-2) which plays a critical role in the production of antigen-specific cytotoxic T cells. Similarly, the production of gamma interferon by self-reactive T cells increases NK cell activity. Finally, B cell stimulating factors, involved in T cell–B cell interactions, can activate self-reactive B cells and initiate the secretion of IgG autoantibody. Such antibodies are directly accessible to target cells and can shorten their life span either by complement mediated cytolysis or cytodigestion by phagocytic cells. Moreover, if the autoantigen is soluble, it can interact with circulating autoantibody to produce circulating immune complexes that can cause a migratory polyarthritis, skin rashes, and other signs and symptoms.

The principal evidence for T cell dependence in autoimmune diseases consists of the following four objectives:

1. The beneficial effect of neonatal thymectomy in animal models of these diseases;
2. A beneficial effect of anti–T cell monoclonal antibodies in some of these diseases;
3. A beneficial effect of anti–IL-2 receptor monoclonal antibodies; and
4. The development of an autoimmune disease similar if not identical to the autoimmune disease in question as a consequence of chronic graft vs. host disease.

The administration of IFN is known to activate T cells and increase the number of NK cells present in the tissues of interest of those treated with the agent.

Table 1. Literative Reports of Interferon-Associated Rheumatoid Arthritis Authors and Reference Number

Authors	Ueno et al	Chazerain et al	Chazerain et al	Chazerain et al	Conlon et al	Conlon et al
Parameters	19	20	20	20	21	21
Cases	One	Two	Three	Four	Five	Six
Age	64	64	52*	42	71	63
Sex	Male	Male	Male	Female	Male	Female
Underlying Disease	Nephroma	CMD	CMD	CAH	LGL Leukem	Melanoma
Dose	9 mu 2 week	3 mu 3 times a week	3 mu 3 times a week	3 mu 3 times	3 mu/day	17 mu 3 times week
Duration of Rx Prior to RA	10 months	6 weeks	6 weeks	6 weeks	9 months	9 months
Pretreatment ANA	—	+	—	—	—	—
Pretreatment RF	—	—	—	+	1:80	—
ANA at Onset of Arthritis	—	++	—	—	—	—
RF at Onset of Arthritis	+++	—	—	+	—	—
Treatment	NSAID steroid DC IFN	NSAID steroid DC IFN	NSAID steroid DC IFN	NSAID steroid DC IFN	NSAID steroid DC IFN	NSAID
Response	Resolved	Resolved	Resolved	Progression	Resolved	Resolved

+* Chronic myeloproliferative disorder
+ Chronic active hepatitis
* Nephrogenic cancer, large granular cell leukemia, melanoma

Thus the development of a putative autoimmune disease such as thyroiditis and/or rheumatoid arthritis is not a totally unexpected consequence of the use of such agents. To our knowledge, however only six cases of RA following IFN therapy have been reported in the English language literature (Table 1).

As shown in the table, only one of the previously described patients continued to have clinically active RA after discontinuing IFN therapy. However, this patient had a prior history of RA and had received a very high dose of IFN (9 mu). Clearly, as discussed above, an exacerbation of preexisting autoimmune disease as a result of proliferation of an existing self-reacting T cell would not be unexpected with IFN therapy. Moreover, it would be unlikely to resolve as a consequence of IFN therapy. Only one of the six cases reported and summarized in Table 1 (Case 1) was negative for RF prior to IFN therapy. Presumably this patient, like ours, became positive for RF as a consequence of IFN therapy. Unlike our patient, this patient's disease resolved with the introduction of NSAIDs and steroids after discontinuing IFN therapy. The present case is unique in that the patient had no history of autoimmune disease, including RA prior to IFN therapy; she seroconverted from RF negative to positive while receiving IFN therapy after 5 months of IFN therapy at a dose utilized to treat viral hepatitis as opposed to cancer, and she developed a newly positive serologic test for RF and ANA. Presumably a new dose of self-reactive T cells directed at the tissue target responsible for rheumatoid arthritis was produced in this patient as a consequence of her IFN treatment. She did not experience clinical resolution of her RA with discontinuation of the IFN therapy. It is likely that once this self-reactive T cell line expanded as a result of the IFN therapy provided, the unique patent endogenous processes that prevent such doses from proliferating were no longer effective, and the disease process produced by them persists.

This case and those summarized in Table 1 suggest that patients being treated with IFN should be screened for the presence of autoimmune disease before and during their IFN therapy, particularly if symptoms of such develop during the course of IFN therapy. In general, it appears as if the prognosis of IFN-induced autoimmune disease is good, with either a resolution or a remission being obtained with discontinuation of IFN. This is not always the case, however, as evidenced by the present case; autoimmune disease initiated as a consequence of IFN therapy may persist despite IFN withdrawal.

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The Authors

Stefano Fagiuoli, MD; Harlan I. Wright, MD; and Judith S. Gavaler, PhD, are staff members at the Oklahoma Transplantation Institute (OTI) and the Hepatic and Digestive Disease Center (HDDC) at Baptist Medical Center of Oklahoma, Oklahoma City.

David H. Van Thiel, MD, is medical director of transplantation at OTI and director of the HDDC.

SPECIAL

Medicaid in Oklahoma

Edward N. Brandt, Jr., MD, PhD; Robert W. Broyles, PhD; Benjamin Demps, Jr., JD; David Falcone, PhD

Medicaid expenditures in Oklahoma increased 98.2% from 1986 through 1991 while those of the United States went up 111.0%. Of this increase, 21.6% was due to inpatient care and 25.9% to nursing home care. With respect to beneficiaries, 34.9% of the increase was attributable to the aged and 31.4% to the disabled. AFDC recipients accounted for only 19.1% of the increase.

TITLE XIX OF THE SOCIAL SECURITY ACT, KNOWN as Medicaid, was enacted in 1965 as a joint federal-state effort designed to pay for health care for recipients of welfare benefits and certain other needy persons. Federal law requires states that receive Medicaid funds to provide health care coverage for certain groups such as families who qualify for Aid to Families with Dependent Children (AFDC), low income pregnant women and young children, and low income aged, blind, and disabled persons including those who qualify for Supplemental Security Income (SSI).

Within federal guidelines, each state is free to design its own program, to determine who will be covered, what services to include, how the program is to be administered, and the level of payments to service providers. The federal government matches state appropriations according to a formula that affords the greatest match to the states with the lowest per capita income although no state receives less than a 50-50 match. In Oklahoma, the federal government pays 70.7¢ of each dollar spent and the state 29.3¢.[1]

This paper reviews the Medicaid program in Oklahoma and compares it to those of other states. Medicaid in Oklahoma is administered currently by the Department of Human Services (DHS), which administers the other welfare programs in the state. As of January 1, 1994, a newly created entity, the Oklahoma Healthcare Authority (OHA), will assume administrative responsibility for the Medicaid program. One of the mandates of the authority is to ensure that all Medicaid recipients are enrolled in a managed care system by 1999. Since Oklahoma will soon alter its approach to Medicaid, it seems important to review the current system and develop the basis for assessing the effects of proposed changes.

Who Receives Medicaid?

The total number of Oklahoma recipients receiving some health care service funded by Medicaid rose from 242,339 in 1986 to 304,659 in 1991, an increase of 25.7% while per capita expenditures rose 53.4%.[2] Examination of Table 1 reveals that the "typical" Medicaid recipient is an adult Caucasian female. Indeed, in 1991, Caucasians made up 68.4% of the recipients and women, 65.6%. Those 65 years old or older comprised only 18.2%, down from 23.5% in 1987. However, the elderly had the highest per capita expenditures of any group, and the per capita expenditures nearly doubled for this age group between 1987 and 1991.

From the Center for Health Policy Research and Development, University of Oklahoma Health Sciences Center, Oklahoma City.

Direct correspondence to Edward N. Brandt, Jr., MD, Center for Health Policy Research and Development, College of Public Health, University of Oklahoma Health Sciences Center, PO Box 26901, Oklahoma City, OK 73190.

What Does Medicaid Cost?

From 1988 through 1991, the national Medicaid program's expenditures have increased dramatically from $51.6 billion to $88.6 billion, with $18.8 billion of that increase in one year (1990-91).[1] The total expenditures by Oklahoma's Medicaid program in 1991 for direct services were $814.3 million, up from $422.3 million in 1986, an increase of 92.8%[2] compared to 111%[3] for the U.S. as a whole.

Table 2 indicates that expenditures for inpatient hospital care and nursing home care accounted for 52.1% of the total expended in 1991, down from 58.1% in 1986, although expenditures for these two categories increased by $178.7 million during that period. The largest percentage increases (452% and 348%) were in outpatient hospital care (from $5.1 million to $28.4 million) and prescribed medication (from $13 million to $58 million). These changes indicate a shift from inpatient to outpatient settings and a greater use or price of medications.

Table 3 contains a summary of expenditures by category of beneficiary. When combined, the aged and disabled accounted for 63.5% of expenditures in 1991, while AFDC recipients accounted for only 25.5%. This finding is consistent with the data in Table 1, which indicate that the aged have a much higher per capita expenditure ($4,807) than any other age group, especially children ($1,048) and young adults ($3,194).

Oklahoma has experienced a particularly dramatic increase in spending on inpatient psychiatric care, with most of this increase occurring for children services. From 1986 through 1991, physician services accounted for only 7.1% of the increase in expenditures, while inpatient care amounted to a 26.2% increase. However, changes in the medical necessity criteria instituted by the Medicaid program in fiscal year 1993 have resulted in a reversal of this trend. Further, declines are anticipated via state legislation that calls for a single point-of-entry gatekeeping case management function to be instituted by the Department of Mental Health and Substance Abuse Services starting in October 1993.

How Does Oklahoma Compare to Other States?

It is difficult to make straightforward comparisons between Oklahoma Medicaid expenditures and those of other states because of differences in eligibility and benefit packages. Further, there are state variations in need. In this regard, it is instructive to note that 15.6% of the Oklahoma population is below the federal poverty line compared to the United States percentage of 13.5%. However, only 8.7% of residents of Oklahoma receive Medicaid compared to 9.6% for the United States.[1] The United States has 16.6% of the non-elderly population uninsured, while Oklahoma has 22.3%.[1] Relative to the national experience, these data indicate that Oklahoma has a larger percentage of people who are in poverty or uninsured.

According to the National Governors Association, eligibility standards in Oklahoma are similar to those of other states[1], and the expenditure per beneficiary in Oklahoma was $2,673 compared to $2,568 for the nation.[1] Hence, Oklahoma differs little from the nation as a whole with respect to per capita Medicaid expenditures, although Oklahoma tends to cover a smaller proportion of the poor population.

From 1987 through 1991, the number of Medicaid recipients in Oklahoma grew from 250,381 to 304,659, an increase of 21.7%[2] compared to a 34.1% increase for the U.S.[3] It is of interest to note that in Oklahoma the expenditure increase from 1986 to 1987 was 18.9% and from 1990 to 1991 was 18.5%, while the comparable increases in beneficiaries were 3.3%

Table 1. Proportions of Recipients

	1987			1991		
	Recipients		Per Capita Expenditure	Recipients		Per Capita Expenditure
	No.	%		No.	%	
Gender						
Male	83,201	33.2	$2,183	104,913	34.4	$2,811
Female	167,179	66.8	1,916	199,746	65.6	2,581
Race						
Cauc.	174,157	69.6	2,249	208,514	68.4	3,019
Black	51,394	20.5	1,407	59,190	19.4	1,863
Indian	19,035	7.6	1,626	27,822	9.1	2,038
Others	5,795	2.3	1,216	9,133	3.0	1,533
Age						
0-5	49,660	19.6	769	68,617	22.5	1,048
6-20	69,941	32.3	1,607	87,933	28.8	1,993
21-64	74,014	28.6	2,541	92,917	30.4	3,194
65-84	43,959	17.3	2,385	39,228	12.9	4,165
85+	15,725	6.2	3,733	16,217	5.3	6,359
Total	250,381	100.0	2,006	304,659	100.0	2,673

and 11.5%, respectively. Hence, the cost per service, the intensity of service, or both must have increased. Indeed, both increased in Oklahoma[2] and in the nation[3].

Table 4 displays a comparison of the effects on expenditures of increased numbers of beneficiaries and per capita expenditures. From 1986 through 1989, per capita expenditures accounted for the majority of expenditure increases. This is a measure of increased cost per service, or the intensity of service, or both. However, that situation changed in 1989 with a marked increase in recipients in both 1989–90 and 1990–91. Most of these increases were due to mandates in federal law that expanded coverage for pregnant women, small children, and the elderly. In spite of an increase of 11.5% in the number of recipients from 1990 to 1991, 34% of the expenditure growth was due to increased service intensity rather than the increased number of recipients. Furthermore, most of the increased numbers of people were children and young adults who are the least expensive recipients.

Table 5 compares the experience of Oklahoma with that of states in the region and with national totals from 1991 to 1992. Note that the United States experienced an increase of 11.7% in the number of recipients and an increase of 11.9% in per capita expenditures; the comparable figures for the region were 16.8% and 21.7%, and for Oklahoma, 18.2% and 3.1%. Hence, Oklahoma experienced a much larger proportional increase in recipients than the nation or the region but a significantly lower increase in spending. Among the states in the region, Oklahoma had the lowest percentage increase in per capita expenditures but the third largest percentage increase

Table 2. Expenditure by Service Category

	Expenditures × 1,000					
	1986		1991		Increase	
Category	Amount	% Total	Amount	% Total	Amount	%
Inpt. Hospital Care	$109,796	26.0	$213,173	26.2	$103,376	94.2
Outpt. Hospital Care	5,137	1.2	28,352	3.5	23,215	451.9
Physician Services	32,900	7.8	57,973	7.2	25,073	76.2
Inpt. Psych Care*	17,013	4.0	44,243	5.4	27,230	160.0
ICF/MR**	64,227	15.2	108,645	13.3	44,418	69.2
Nursing Home Care	135,569	32.1	210,921	25.9	75,352	55.6
Prescribed Medication	12,987	3.1	58,134	7.1	45,147	347.6
All Other	44,695	10.6	92,834	11.4	48,139	12.3
Total	422,324		814,275		391,951	92.8

* Includes only inpatient care in a psychiatric hospital. Inpatient psychiatric care delivered in all other hospitals is included in inpatient hospital care.
** ICF/MR = .

Table 3. Expenditure by Beneficiary Category

	Expenditures × 1,000					
	1986		1991		Increase	
Category	Amount	% Total	Amount	% Total	Amount	%
Aged	$161,964	38.3	$266,163	32.7	$104,199	64.3
Disabled	134,627	31.9	250,820	30.8	145,459	108.1
AFDC-Adult	44,141	10.5	61,839	7.6	17,798	40.3
AFDC-Child	72,725	17.2	145,862	17.9	73,137	100.6
All Others	8,867	2.1	89,591	11.0	80,724	910.4
Total	422,324		814,275		391,951	92.8

Table 4. Expenditure Increases

Years	Proportional Effects of Recipients	Per Capita Expenditures	Recipients Increase	Expenditure Increase (x 1000)
1986-87	0.18	0.80	8,042	$79,859
1987-88	–0.15	1.18	–5,761	79,271
1988-89	0.38	0.60	8.483	53,150
1989-90	0.96	0.04	20,152	52,928
1990-91	0.62	0.34	31,404	126,742

Table 5. Regional Comparisons 1991-1992

State	1991 Recipients	1991 Per Capita Expenditure	1992 Recipients	1992 Per Capita Expenditure	% Increase Recipients	% Increase Per Capita Expenditures
Arizona	313,142	$2,720	402,212	$3,065	28.4	12.7
Arkansas	284,674	2,645	320,875	2,988	12.7	12.9
Colorado	223,444	3,481	258,690	3,958	15.7	13.7
Kansas	209,329	3,367	226,991	4,136	8.4	22.8
Missouri	503,310	3,375	554,477	4,263	10.2	26.3
New Mexico	161,995	2,413	211,805	2,549	30.7	5.6
Texas	1,228,629	2,446	2,024,554	3,114	17.1	27.3
Overall	3,424,523	2,747	3,999,604	3,342	16.8	21.7
Oklahoma	304,659	3,013	360,039	3,105	18.2	3.1
United States	28,279,781	3,342	31,594,047	3,740	11.7	11.9

in recipients. Texas and Missouri experienced the largest percentage increase in per capita expenditures, while New Mexico and Arizona showed the greatest increase in recipients. Hence, the experience of Oklahoma is consistent with the view that the Oklahoma Medicaid program has been relatively effective in controlling costs.

Summary

From 1986 through 1991, national expenditures by Medicaid increased at a dramatic rate (111.0%), as did those in Oklahoma (92.8%). For the U.S., 34.1% of this increase was due to increased enrollment, 31% to medical inflation, and the remainder to increased utilization.[3] Oklahoma differs somewhat in that we experienced only a 21.7% increase in enrollment,[2] with the rest of the increase due to the other two factors. However, it should be noted that recipients in Oklahoma over the age of 65 decreased by 7.0%,[2] while in the nation this age group increased by 4.2%.[3] This age group is by far the most expensive, and that is a potential explanation of the Oklahoma differences.

These data should dispel some preconceptions about Medicaid recipients in Oklahoma. For example: (1) nearly two-thirds of the recipients are female; (2) 68% are Caucasian; and (3) 18% are over age 65, but they account for 33% of expenditures. It is also important to emphasize that Medicaid does not cover all of the poor. For example, although 15.6% of Oklahomans are below the poverty line, less than 10% are eligible for Medicaid.

References

1. "Medicaid at the Crossroads," Kaiser Commission on the Future of Medicaid.
2. "Medicaid Cost Study," Center for Health Policy Research and Development, University of Oklahoma Health Sciences Center.
3. "The Medicaid Cost Explosion: Causes and Consequences," Kaiser Commission on the Future of Medicaid, 1992.

The Authors

Edward N. Brandt, Jr., MD, PhD, is a professor in the Department of Health Administration and Policy, College of Public Health, and co-director of the Center for Health Policy Research and Development at the University of Oklahoma Health Sciences Center (OUHSC) in Oklahoma City.

Robert W. Broyles, PhD, is a professor in the OUHSC Department of Health Administration and Policy at OUHSC and a member of the Center for Health Policy Research and Development.

Benjamin Demps, Jr., JD, is cabinet secretary of Health and Human Services and director of the Oklahoma State Department of Human Services.

David Falcone, PhD, is professor and chairman of the OUHSC Department of Health Administration and Policy.

NEWS

OU College of Medicine makes *U.S. News & World Report* Top 20

For the second consecutive year, the University of Oklahoma College of Medicine was ranked among the top 20 comprehensive medical schools in the country, according to the March 21 issue of *U.S News & World Report*. The news magazine publishes a cover article each spring citing the nation's best graduate schools in law, business, medicine, and engineering.

Because medical schools educate medical students, residents, and practicing physicians, as well as perform research, the *U.S. News* editors opted to divide the nation's 126 medical schools into two categories—those more oriented toward research and those more oriented toward training primary care physicians. The OU College of Medicine falls into the latter category, and was named 20th among the nation's best comprehensive medical colleges.

"Despite uncertainties about the directions that medical education and health care will be taking with ongoing reform measures, our college's applications are at an all-time high, and we have a higher-than-average number of students who are entering primary care fields, including family medicine," notes Dr. Douglas Voth, executive dean of the OU College of Medicine. "I think we are seeing an increase of more altruistic applicants—students who enter medicine because of a strong desire to take care of patients and contribute meaningfully to community life, rather than just opting for a secure financial future and a comfortable lifestyle."

Based upon recent information on location and career selection gleaned from the college's 1994 graduates, 59 percent are entering generalist (primary care) residency programs, and 49 percent are staying in Oklahoma for further education and training, Voth adds.

The OU College of Medicine also boasts the nation's highest Native American student enrollment, with 58 new applicants last year. In all, one-third of the college's applicants are minority students.

"In addition to continuing our recruitment of minority students, we are developing new measures to attract students into general internal medicine, pediatrics, family medicine, and obstetrics—what is known as 'primary care,' and to attract our resident graduates into practice in rural Oklahoma," Voth says. "There is a substantial shortage of this type of care in Oklahoma, and the demand is expected to increase under various legislative and administrative reforms. We have several programs in place and others being developed to encourage applicants who are considering these disciplines."

Health Care Quality Improvement Project

Stephen M. Spielman, MD, Principal Clinical Coordinator, OFPR

On October 1, 1993, the Oklahoma Foundation for Peer Review (OFPR) embarked on implementation of the 4th Scope of Work as outlined by the Health Care Financing Administration (HCFA). Primary in this effort is the development of the new Health Care Quality Improvement Project which drastically changes the emphasis of peer review organization (PRO) activities. I feel it would be safe to say the focus of previous PRO activities often was regarded by those practicing in the medical community as police-like actions including a punitive intent. The new program is clearly intended to be of an educational nature and we sincerely hope here at the OFPR that it is perceived in this way.

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Health Department

Infants with Sickle Cell Disease Need Early Care

Early diagnosis of infants with sickle cell disease and treatment with penicillin prophylaxis in the first two months of life can reduce the number of deaths from sepsis. This was the finding of a National Institutes of Health consensus panel that recommended all newborns, "regardless of race," be screened for sickle cell disease to reduce morbidity and mortality." Consistent with this consensus statement and the recommendations of the American Academy of Pediatrics, the Oklahoma State Board of Health, in 1991, adopted regulations specifying newborn screening for all Oklahoma infants for sickle cell disease. Newborn screening data from 1992 indicate that eleven infants were born with sickle cell disease and 433 with sickle cell trait.

"Sickle cell disease" describes a group of genetic disorders characterized primarily by hemoglobin S (Hb S). The most common type of sickle cell disease, and usually the most severe, is sickle cell anemia (Hb SS) which has an incidence of 1 in 375 black births. Other forms of sickle cell disease which may vary in severity include hemoglobin SC disease (incidence 1 in 835 black births), and hemoglobin S beta thalassemia (incidence 1 in 1,667 black births). Although these diseases are most common in persons of African, Hispanic, Caribbean, South and Central American, Middle Eastern, East Indian, and Mediterranean ancestry—sickle cell disease can occur in any racial group.

Clinical Practice Guidelines for sickle cell disease were published by the U.S. Agency of Health Care Policy and Research in April 1993. Included are guidelines for screening, laboratory methods, medical management, and counseling for families. Copies of these guidelines are available through the Oklahoma State Department of Health Chronic Disease Service, (405) 271-4072, and the OSDH Maternal and Infant Health Service, (405) 271-6617. The Maternal and Infant Health Service provides clinical services for infants and children with sickle cell disease through comprehensive sickle cell clinics at Children's Hospital of Oklahoma in Oklahoma City, (405) 271-5311, and the Abnormal Hemoglobin Clinic of the University of Oklahoma Health Sciences Center in Tulsa, (918) 838-4720 or (918) 838-4823. Services provided through these clinics include medical care, patient education, and case management. Families with sickle cell trait may be referred for counseling to the Oklahoma Chapter of the National Association for Sickle Cell Disease, (918) 428-1974.

OFPR *(continued)*

With the new direction upon which we are embarking, we do feel the PRO can do more to improve the quality of care and the cost effectiveness of health care by bringing typical care into line with best practices rather than by inspecting to identify erred treatment in individual cases as has been the PRO's focus in the past. The mechanisms utilized to assess quality of care in the past and subsequent methods to improve quality when it was felt to be indicated often lead to adversarial relationships and, occasionally, to outright hostility. Perhaps more important, however, was the lack of any way to measure overall improvement in health care delivery. The new process has built into it a measure of improvement which will be a major focus of activities as they go forth.

The process we are presently initiating will not be entirely foreign to physicians actively practicing on most hospital staffs. It will consist of quality improvement projects quite similar to those performed within your institutions as mandated by the Joint Commission on Accreditation of Health Care Organizations. The advantage of the PRO performing such activities, rather than leaving them to each individual institution, rests in the assessment of pattern analysis of care and outcomes of care between like providers. *Provider,* in this case, will refer to hospitals or institutions and not to individual physician practice. There is support for this approach in the literature, often termed "variations research," which reveals significant differences in both processes and outcomes of care between institutions similar in size, geographic location, and services rendered. We plan to work with the local health care community to identify and interpret scientifically sound

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WORTH REPEATING

Medicine still great profession says TCMS president

As I have observed the charges that have been taking place in medicine over the last couple of years, I have often wondered what effect these changes will have on the attitude of physicians (including my own) regarding our profession and the future of that profession. This attitude was brought home to me recently when I heard a medical student discussing his contacts with his teachers and some of the older men in medicine. The student felt that the field of medicine was challenging, that medical school had been interesting, and he was enjoying the contact with the professors as well as the patients. However, as part of the interaction with his teachers he felt that there was a negative attitude. In "after rounds discussions" the conversations often centered on malpractice, DRUGS, decrease in salaries, medicare fees and paperwork, health reform, etc. He had heard physicians on several occasions state that "medicine was not fun anymore." They talked about the good old days and how things would never be the same again.

It was not so unreasonable for me to suggest that the positives of the career outweigh the negatives. Of course there are problems, but we ought to be among these seeking solutions to those problems. We should not lose sight of how lucky we are to have a profession in which we do well for ourselves by doing well for others.

The satisfaction of relieving pain, restoring function, solving chemical problems, etc., will remain the essence of doctoring, no matter what changes we see in the organizational and economic structures of medicine. Despite the increase in bureaucratic controls, doctors still retain a degree of autonomy rarely found in other professions. Medicine is still a great profession, held in high esteem by most of the public.

Of course there are concerns. If we are going to help reverse the trends and keep some control of medicine in general and of our practices, we need to mobilize our natural allies—our patients and the public at large. They have the greatest stake in preserving excellence in medical care. If we focus on our primary responsibility to serve as advocates for our patients, we will maintain our integrity and provide leadership for a broad group of the public in defense of health care.

We have an obligation to sustain the highest goals of our medical students and those considering medicine as a future profession. Students certainly need to know about the problems; but let's help them keep these problems in perspective. Let's enlist our students in the campaign for quality in medical care. To succeed in the future and to keep medicine where

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OFPR *(continued)*

parameters of practice to measure the quality of care at each provider hospital. This will enable us to determine "benchmark" or "best" practices as well as those which have room for improvement in any particular area. Subsequently, through an educational effort, it is then hoped that all care can be brought as close as possible to the level of our benchmark practices. Improvement plans put in place to effect this change will be directed by each individual institution and will not be forthcoming from a federal level nor from the local PRO itself. The local PRO would be available, however, to assist in any improvement activities if so requested.

Though the individual chart review of the past is not disappearing entirely, noting a few numbers does emphasize the degree of the present change. A few years ago there was a random sampling of 50% of Medicare beneficiary charts. Currently this has fallen to 6 to 8% and is likely to fall further in the near future. Whether this will disappear entirely is not clear at this time.

I think it is quite obvious that for the PRO to perform in this manner it must be able to address issues in a scientifically sound manner. We have assembled a team of players for this effort with strong statistical support from Dr. Cynthia Murray, a biostatistician who has been involved in this area for many years at the University of Oklahoma Health Sciences Center. Utilizing this expertise, along with input from appropriate individuals regarding the practicing physician's and nurse's points of view, we will subsequently embark on a systematic analysis of a national data base provided to us. This data base is derived from the UB92, which in simpler terms is the front

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WORTH REPEATING (CONTINUED)

it is today as a profession, we will need the efforts of the best and the brightest.

What we do as doctors, most of the time, is quite gratifying. I cannot imagine a more interesting or more gratifying or more challenging career. Let's make sure our patients and our students get that message from us.

—Boyd O. Whitlock, MD
President
Tulsa County Medical Society

Reprinted by permission. *Tulsa Medicine*, March 1994.

PLICO shines in wake of national incident

To the Editor: The *Business Week* article about a "cheap" malpractice offering that went awry for many a doctor is another example of why the Oklahoma product through PLICO has such a great value. Although malpractice insurance articles aren't a usual component of our [Oklahoma County Medical Society] *Bulletin,* I would suggest that some synopsis (below) of this article might be of interest within the *Bulletin.*

Oklahoma physicians might be interested in hearing about a malpractice fiasco that affected numerous physicians and other health professionals across the country. A report in the March 7, 1994, *Business Week* outlines how members of a Maryland family (the Bramsons) searched for prospective customers through purchases of lists of high risk medical specialists; they also sold group malpractice policies to associations representing non-physician groups. Once getting such customers on board, they simply collected the premiums, and then, when court judgments were levied, used a system of loophole invocations and termination of coverage. this type of "escapism" would seem easy enough to stop, but the Bramsons apparently stayed ahead of regulators by shifting their business among as many as 53 companies, and a host of mail drops and phone numbers.

The *Business Week* story highlights the laments of one California physician who lost about $100K in premiums over a 7-year period, and then ended up having to remortgage his house and spend his children's college funds. Although this ring was broken (you've got to read the *Business Week* story to see how the case was first cracked), there's little money to cover all the outstanding claims.

What a story! And what another reason to be thankful we have the "good people" at PLICO.

—Jerry R. Hordinsky, MD
Oklahoma City

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OFPR (continued)

sheet on each chart, upon which each diagnosis and procedure is listed and coded. This information will be useful in itself as feedback to providers but also to a large extent will be utilized to create focused chart abstraction projects related to well accepted standards of care as practiced in our state. I am quite hopeful at this time that the approaches we are taking in our utilization and assessment of such data are sound and will be beneficial when presented to involved institutions and their medical staffs.

The backbone of the new program is known as the "local cooperative project." Of these projects, approximately half will involve issues of quality of care and the other half will be divided among coding, documentation, and utilization issues. Obviously many projects will overlap in these areas. I think it might be helpful at this time to give you a basic overview as to how this system will function. There are 117 providers in the state of Oklahoma. I would anticipate nine to ten local cooperative projects to be ongoing at any given time. Each project will likely include five to ten institutions, though conceivably this could range anywhere from one to twenty, and in a rare instance a project could involve all institutions. Again, I wish to emphasize that the derivation of the vast majority of these projects is local in nature and is not mandated by HCFA as to the specific areas evaluated. In the future, however, it is clear there will be national projects, though it appears that this is likely to be relatively small in number at any given time.

Project ideas may be conceived or come from any source including the literature, individual institutions, and physicians. Projects performed in other states that appear to be successful will also be considered. I would welcome ideas from any source at any time and would be glad to discuss them with you.

I feel it would be helpful to describe the devel-

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OFPR *(continued)*

opment of a project in the temporal aspects as well. Following conception of a project idea, the mechanical activities of developing a specific hypothesis, a focused abstraction tool with which to gather data from individual charts, and the methods of statistical analysis will be undertaken by the PRO staff. Subsequently, two advisory committees will review the project before it is actually initiated. Most important to any specific project will be an ad hoc committee, termed a "study group." This will often consist of physicians from the institutions to be involved in that particular project, with a specific intent to obtain not only the appropriate specialist but also to include family practitioners and general internists. The purpose of this study group will be to assess the appropriateness of the project both in its basic concept and in the validity of its design. At this time members of this group will also be able to express any concerns regarding the project as related to their specific institution. It is hoped these projects will be truly collaborative efforts between the PRO and the medical community. Additionally,the PRO has developed a Master Committee that includes eleven physician members as well as six non-physician members. The physicians include those from the major specialty groups, including family practice, as well as representatives from each of the academic institutions in our state. The non-physician members include a representative from the hospital association, the nursing association, two hospital administrators, and also two Medicare beneficiaries. The Master Committee will function as an advisory board in regards to any concerns relating to any of the projects.

Considerable thought has been given as to how institutions will be selected for various projects. I suspect much of the selection will be random in nature but there will be some attempt to select a reasonable distribution of urban versus rural institutions along with geographic considerations. Obviously to compare activities in various areas, hospitals with similar services will have to be com-

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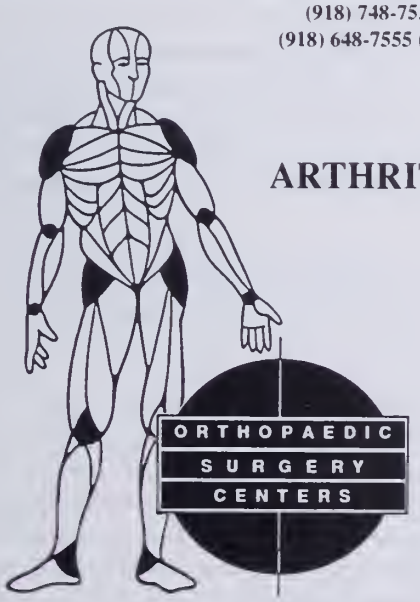

ARTHRITIS SURGERY CENTER OF OKLAHOMA

Norman L. Dunitz, M.D. — Mayo Clinic trained; Oklahoma pioneer in Joint Replacement development

Scott J. Dunitz, M.D. — Mayo Clinic trained; specializing in Joint Replacement Surgery and Surgery of Arthritis

James C. Slater, M.D. — Campbell Clinic trained; Full Fellowship Specialty in Arthritis Surgery; Harvard/Tufts

Readers share editor's views on state's medical licensing board

To the Editor: I could not agree more with your editorial ["Still an Awesome Duty"] in the March issue of the JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION.

I hope that you are doing all right and I appreciate all of the time that you have given for the benefit of your colleagues.

—B.J. Rutledge, MD
Oklahoma City

To the Editor: Your editorial in the March issue of the OSMA JOURNAL is appreciated, as well as well written—as are all of your editorials. I know two of the physicians on the Board well, and I highly respect them and trust their judgment. However, I do share your impression that decisions may be swayed by interpretations of investigators, administrators, and attorneys.

Having had no direct experience with the Board (fortunately), I agree that their activities are not similar to physician peer review (with which I have considerable experience).

I also agree strongly with your opposition to HB 2123. Mandated CME has been shown to be unfair, unrealistic and, most importantly, ineffective in improving the quality of patient care. The Board of Medical Licensure would seem to be the least qualified to direct such CME requirements.

I also agree with your concerns regarding the compensation and length of service of the Board members.

Thanks again for a fine editorial, and keep up the good work.

—Warren L. Felton II, MD
Oklahoma City

Letters should be addressed: To the Editor, OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118.

OFPR *(continued)*

pared. Some projects may have selection based on the Medicare Hospital Information Project (MHIP). As a matter of review, this is a statistical analysis from the Part A Medicare claims data base which yields mortality data in relationship to the major diagnostic codes and procedures. Also, in many instances, it may be appropriate to merely take volunteers for projects in which particular institutions have an interest.

I doubt many would disagree that, in some form, peer review is here to stay, and it is with cautious optimism that we here at the OFPR embark upon this new effort which is truly meant to be educational in nature. The initiative itself a cooperative effort by design, and its degree of success will relate in great part to the degree of collaboration that occurs. Providing the best quality of health care to our patients is certainly one goal that we all have in common, and I sincerely hope that the OFPR can serve as a successful facilitator in the design and interpretation of this new program of statistical pattern analysis and educational efforts.

Helping to support OSMA

Remember your meeting exhibitors

A number of exhibitors and contributors played a major role in ensuring the success of last month's OSMA Annual Meeting at Shangri-La. Some are OSMA supporters of long standing and some are new this year; all were appreciated for their participation, particularly at this year's out-of-the-way venue:

ABC Home Health Services, Inc.
Administrative Medical Office Services, Inc.
Administrative Service Corporation
American Home Health Services, Inc.
American Transitional Hospital
Baylor Institute for Rehabilitation
Bank of Oklahoma
Benchmark Motors
Boots Pharmaceuticals
C.L. Frates and Company
Guaranty Bank & Trust Co.
Harrison Associates, Inc.
Home Health Care Associates
I.C. System, Inc.

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Dr. Garth Splinter to head Oklahoma Health Care Authority

Garth Splinter, MD, MBA, a nationally recognized leader in health care reform, has been appointed administrator of the Oklahoma Health Care Authority. Governor David Walters announced March 16.

Governor David Walters announced the appointment March 16. "His credentials as a physician and his educational background in business administration make him uniquely qualified to serve as administrator of the Oklahoma Health Care Authority," the Governor said.

Senate confirmation of the appointment is required and Sen. Bernest (D-Oklahoma City) will carry the appointment. "Dr. Splinter has certainly been helpful in Oklahoma health care policy in the past, and I expect him to be a real leader in this new post," said Sen. Cain.

In January 1991, Dr. Splinter was appointed as the Governor's special advisor on health care issues and was a key figure in the development of Governor Walters' Oklahoma Family Choice Health Plan.

He is currently director of the health affairs and rural health programs at the University of Oklahoma Health Sciences Center and assistant commissioner for health care reform at the Oklahoma State Department of Health. He chaired the Commission on Oklahoma Health Care and was a member of the Basic Health Benefits Board from March 1992 to January 1993.

He previously served as medical director with Oklahoma State and Education Employees Group Insurance Board and as assistant professor/director of planning and development with the University of Oklahoma College of Medicine.

Dr. Splinter was graduated from the Harvard Graduate School of Business Administration with a masters degree in business administration in 1976. He earned his medical degree from the University of Oklahoma College of Medicine in 1984.

He is a member of the American Academy of Family Physicians, American Medical Association, Oklahoma State Medical Association, Oklahoma Academy of Family Physicians, and American College of Physician Executives.

Dr. Splinter and his wife, Michele, have three children and make their home in Oklahoma City.

Exhibitors *(continued)*

Innovative Physician Services
Laureate Psychiatric Clinic and Hospital
McNeil Pharmaceutical
Medaphis Corporation
Medical Arts Laboratory
Medical Imaging Center of Oklahoma
Miles Pharmaceuticals
NEO Rehabilitation Hospital
Oklahoma Association for Home Care
Oklahoma State and Education Employees Group Insurance Board
Pfizer Labs
Physician Manpower Training Commission
Presbyterian Hospital
Professional Office Management
P&P Industries/Raley Pharmacy
Southern Medical Association
Stillwater National Bank
University of Oklahoma Health Sciences Center Libraries
Works & Lentz, Inc., Attorneys-at-Law

Toxicology report lists ten most common sources of poisonings

The ten most common poisoning exposures based on a 10-year study of more than 12.5 million accidental or intentional poisonings are reported in *Clinical Toxicology Forum,* Vol. 5, No. 5. The report, gathered from information from the 1993 Annual Report of the American Association of Poison Control Centers, listed the following as the most common sources of poisoning:

1. Cleaning Products. Household cleaning products are the number one source of poisonings in the U.S. More than 60 percent of the exposures occur in children because cleaning agents are so readily available. The most dangerous of these products are corrosives, such as drain cleaners and toilet bowel cleaners, bleach, and soap and detergents.

2. Analgesics. Analgesics constitute not only the second most common poisoning exposure, but also the second most lethal. Nearly 50 percent of poisonings in this category involve acetaminophen-containing

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Poisoning sources *(continued)*

products. Salicylate, ibuprofen, and narcotics are also included in this category.

3. Cosmetics. Cosmetic exposures are very common but relatively harmless. The majority of poisonings in this category involve children who copy the actions of adults. Mouthwashes and perfumes contain enough alcohol to poison a child.

4. Plants. Plants have been incorrectly blamed for more poisonings than they actually cause. However, some plants are dangerous and life-threatening if eaten. If there's a doubt, call the Poison Control Center. Another good idea is to know by name all plants and shrubs around your home.

5. Cough and Cold Preparations. All analgesics, antihistamines, cough suppressants, decongestants, and expectorants have the potential to poison. They should always be used with caution and kept out of the reach of children.

6. Hydrocarbons. The most common source of hydrocarbon exposure is gasoline fumes. Hydrocarbons such as gasoline, charcoal lighter fluids, or lamp oil can be deadly if aspirated into the lungs.

7. Bites and Stings. Of 471,000 bites reported during a ten-year period, only 21 resulted in death. The largest number of fatal reactions, 48 percent, were to bee stings. Most other bites, including bites from snakes and spiders, did not cause death, but more than 50 percent of the victims did suffer minor to moderate toxic reactions.

8. Topicals. Topical drug products are also common sources of exposure in both adults and children. Adults sometimes mistake the packaging for toothpaste or otherwise consume the product inadvertently.

9. Foreign Bodies. Foreign body exposures usually occur in children,who may swallow coins, parts of toys, or other small items. The principal concerns are aspiration of the object or obstruction of the esophagus. However, poisonings can occur with disc-type batteries, lead items, and coins.

10. Chemicals. Cyanide and ethylene glycol are the most common culprits. Not only are these types of chemicals on the top 10 most common list, they also are eighth on the most lethal poisoning list.

If poisoning is suspected, call the Poison Control Center at 1-800-522-4611 statewide or (405) 271-5454 in the Oklahoma City metro area.

In Memoriam

1993	
William Finis Ewing, Jr., MD	March 31
John Robert McInnis, MD	March 31
Chester Randall Seba, MD	April 4
Edward Cotter Murray, MD	April 9
Eric McLain White, MD	April 23
John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17
1994	
Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13
Earl Mathiews Woodson, MD	February 20
Orville Main Rippy, MD	March 11
Minor Elliott Gordon, MD	March 14

Deaths

Minor Elliott Gordon, MD
1914 - 1994

OSMA Life Member Minor E. Gordon, MD, Claremore native and son of an early day Claremore physician, died March 14, 1994. Dr. Minor, a long-time general practitioner in his hometown, earned his medical degree at the University of Arkansas in 1942. During World War II he served with the U.S. Army in the Pacific, earning the rank of captain, and by 1946 he had returned to Claremore to practice. He delivered more than 3,000 babies during his career, before retiring in 1986.

(continued)

DEATHS (CONTINUED)

Orville Main Rippy, MD
1913 - 1994

Seminole native Orville M. Rippy, MD, died March 11, 1994, in Stillwater. Dr. Rippy, an OSMA Life Member, earned his medical degree at the University of Oklahoma School of Medicine in 1939. The general practitioner served on active duty with the U.S. Army during World War II. At the time of his discharge in 1946, he had earned the rank of major. Dr. Rippy lived briefly in both Shawnee and Seminole before entering the service. He established a practice in Stillwater in 1946, which he continued until his retirement in 1984.

Earl Mathiews Woodson, MD
1897 - 1994

Earl M. Woodson, MD, a Poteau physician for some 65 years, died February 20, 1994. Dr. Woodson was born in Monroe, Indian Territory. After earning his medical degree at Tulane University in 1922 and serving a few months in the U.S. Army during World War I, he returned to Poteau to enter a general practice with his father, Dr. Benjamin D. Woodson. In 1973, Dr. Woodson was awarded a 50-year pin and Life Membership from the OSMA.

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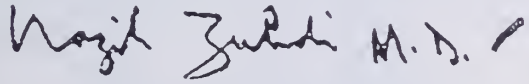

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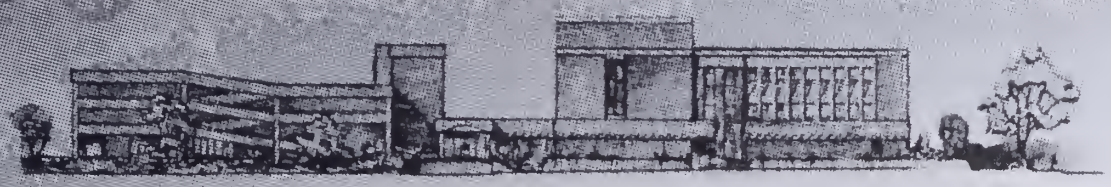

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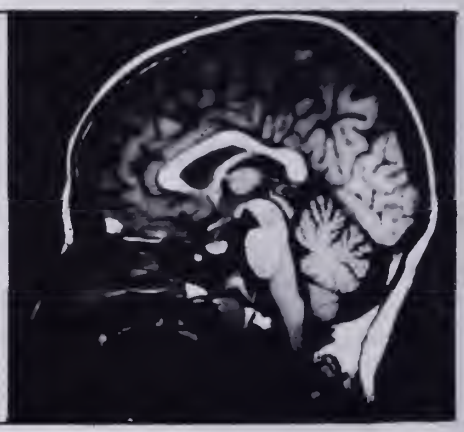

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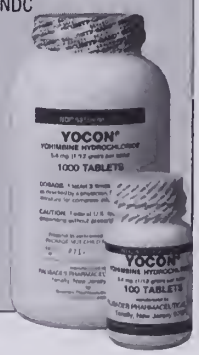

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3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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ALLIANCE

From Auxiliary to Alliance

By the time this JOURNAL goes to print, the organized group once known as the Oklahoma State Medical Association Auxiliary will have a new name. As of the OSMAA House of Delegates meeting on April 15th, 1994, our name officially changed to the Oklahoma State Medical Association Alliance. Our national organization changed its name to reflect the word *alliance* at the national convention held in June 1993. Our county organizations had the option of using either *auxiliary* or *alliance.* In Oklahoma some did and others did not change. More important, our goals and purpose have not changed. We are ever committed to our local community needs, to our AMA-ERF projects, and to intensifying our health promotions against family violence.

We are living in an exciting medical time. Our physicians may seem like they are in the battle alone, but the Alliance, under the leadership of Sherry Strebel, our State Legislation Chairman, will continue to provide the education to enable Alliance members to be better informed on the current status of health system reform. We will be working in our communities to get our side of the issue heard.

I am in awe of the work that the auxilians have accomplished across our nation. We have raised more than two and a half million dollars in the last year for the American Medical Association Education and Research Foundation (AMA-ERF). We have raised close to that amount in support of local health programs, and auxilians gave more than $500,000 in health career scholarships and loans to students.

When it comes to legislative action, our achievements are equally impressive. Last year voter registration projects in 28 states assured that the medical community made its voice heard at the polls. We have contact systems and telephone banks that enable members to respond quickly to state and federal legislation affecting physicians' practices.

We are fortunate to have wonderful national confluences sponsored by the American Medical Association Alliance. Each year we ask our county president-elects to participate in these leadership workshops. We will continue a program that Karen Mask, our immediate past OSMAA president, initiated. Many of our smaller groups cannot financially afford the expense of sending a representative to our national meetings. Instead, members of the state executive board, our Membership Chairman, our AMA-ERF Chairman, and our Health Promotion Chairman, as well as our president-elect will join me this summer to present the Pony Express across the state. The Executive Committee feels that we will broaden the knowledge base of our county officers and let them know that the state Alliance is always ready to assist them. The best part of the Pony Express is the opportunity to make many new Oklahoma friends!

—Maggie Hubner
OSMAA President

THE LAST WORD

■ **The University of Oklahoma College of Medicine** Alumni Association held its annual awards dinner Friday, April 15, in conjunction with the OSMA Annual Meeting at Shangri-La Resort, Afton, Oklahoma. Honored as Private Practice Physician of the Year was John M. Huser, Jr., MD, a family practitioner in Weatherford. Fred G. Silva, MD, Oklahoma City, was named Academic Medicine Physician of the Year for his work in anatomic and clinical pathology. The Amicus Medicinae "Friend of Medicine" recipient was Oklahoma City civic leader Stanton L. Young.

■ **The Oklahoma State Medical Association's** MCO and IPA temporary boards of incorporators met April 5 in Oklahoma City, their last meeting prior to the Annual Meeting of the OSMA House of Delegates at Shangri-La. Chaired by Dr. G. Lance Miller, Tulsa, and Dr. Jay A. Gregory, Muskogee, the boards worked out final details of organizational, legal, financial, and marketing information requested by the House of Delegates during its special session February 27. In addition to Drs. Miller and Gregory, the following physicians served on the boards:

MCO Board—Donald E. Crawley, Stillwater; Joe S. Hester, Muskogee; Larry L. Long, Oklahoma City; Dennis E. Mourning, Claremore; Michael L. Winzenread, Oklahoma City; C. Terrence Dolan, Tulsa; James S. Jones, Duncan; Mary Anne McCaffree, Oklahoma City; Floyd F. Miller, Tulsa; David M. Selby, Enid; Kenneth W. Whittington, Bethany; Eldon V. Gibson, Shawnee; Perry A. Lambird, Oklahoma City; William C. McCurdy III, Norman; Philip Mosca, Oklahoma City; and Richard Bruce Winters, Poteau.

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■ **Tulsa dermatologist Steven A. Smith, MD, has** developed a drug for the treatment of psoriasis. Psorizide caused no side effects during initial testing, Dr. Smith says. Approval of Psorizide by the Food and Drug Administration (FDA) is pending.

■ **Rocky M. Morgan, MD, house staff; E. Montez** Mutzig, MD, full-time faculty; and Keith L. Stanley, MD, clinical faculty are winners of the 1994 Aesculapian Award from the University of Oklahoma College of Medicine, Tulsa, Class of 1994. Each year's graduating class presents the award to individuals who exemplify excellence in teaching the art and science of medicine.

■ **John B. Nettles, MD, Tulsa, has received the** Graham Waite Award from the American College of Obstetrics and Gynecology (ACOG), District VII. The award recognizes Dr. Nettles' leadership and expertise in the provision of women's health care in Venice and Vienna.

■ **The five most commonly reported diseases in** Oklahoma in 1993 were campylobacter, giardiasis, salmonellosis, shigellosis, and hepatitis A, according to the Oklahoma State Department of Health. The department also notes the five most common diseases in 1917: diphtheria, scarlet fever, smallpox, measles, and typhoid fever.

■ **OSMA student members Diane M. Snyder, MS** III, and Kelley A. Burst-Singer, MS IV, Tulsa, are cofounders of the newly formed student chapter of the American Women's Medical Association. Their goals this year are to create a big sis/little sis support system for incoming MS IIIs and to arrange mentorship dinners with women physicians in the Tulsa area. They also hope to send several members to the regional AMWA meeting this month.

■ **A *Wall Street Journal*/NBC News poll of 1,500** adults across the country indicates the public has greater confidence in the AMA's ideas and proposals on health system reform than in President Clinton's. The AMA also scored higher than major health insurance companies, hospital groups, labor unions, the National Association of Manufacturer, and the U.S. Chamber of Commerce. The poll also found that Americans consider physicians the most important source of information when making medical/health care decisions, according to the report in the AMA's March 28 *This Week* newsletter.

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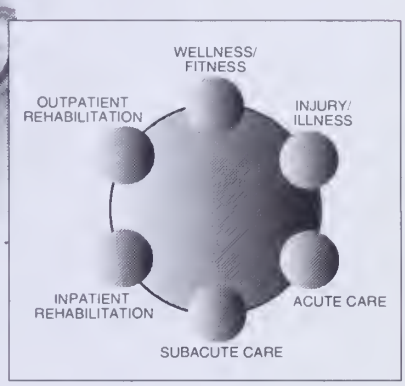

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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"Leaves of three, let it be." Innocuous-looking poison ivy waits in ambush for the unwary.

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EDITORIAL

Ray V. McIntyre, MD

A New Direction

Opportunity knocks, dressed for work and pressing an insistent time schedule, and it is the chance afforded to Oklahoma physicians in the formation of a health insurance company by the OSMA House of Delegates. In this year of health reformation, Oklahoma physicians may have an opportunity to prevent the engulfment of the remainder of our patients in the socialistic quagmire of government medicine. The hard work and time sacrifice of many physicians can save many citizens from that unhealthy status.

The appointed IPA and MCO boards represent a brilliant pool of medical talent, and these physicians must now learn, while actively practicing medicine, to also run a complex fiduciary enterprise with a unique esoterica. The venerable task of these boards now is to marry corporate commerce with scientific healing, while preserving the medical freedom of American citizens. This Herculean task must be done in the interstices of medical practice, for a successful wedding of business and healing can only be accomplished by practicing; the task cannot be delegated to insurance people or medical administrators.

These boards must construct a ship to sail—like Columbus' *Santa Maria*—into unknown waters. The future is always uncertain, but there are sure to be storms generated by the politicians, bureaucrats, and insurance executives, as none of these types have much desire to see any physician success in this ocean. Also, the ship must be constructed to grow and to change, for many of the conditions necessitating the enterprise are temporary crises that will fade away with time. For example, the managed care organization concept is a jury-rigged economic checkvalve that must sometime be replaced by patient-controlled requests for service. Numerous other similar quandaries now exist and will require resolution by physicians who understand the nuances of the needs of the sick.

The greatest asset of the medical profession in this upcoming renovation is the stark fact that medical care is now an essential commodity. Some word-slingers try to deny that medical care is a commodity, but the reality is that *any* medical care costs something, and always will. And the physician's time, made significant by medical knowledge, is of concrete value to the sick patient. Thus the citizens *need* physician service and, despite the efforts of a jillion ancillary practitioners to get a piece of the action, the physician remains the central source of a personal service essential to our way of life.

For the essentiality of medicine to be a constructive force in our enterprise, physicians must consciously develop the unity of the profession. While different specialties and different regions often have differing priorities and worries, we must constructively emphasize those goals and values common to all physicians. All physicians want to deliver good quality medical care to all their patients, and every specialty has some special asset to contribute to patient care. The health of the patient must be the lodestar of the system, and all other considerations must be made ancillary.

If the majority of patients receive the kind of medical care that we want our own families to receive, the enterprise will be a success.

Ray V. McIntyre, M.D.

President's Page

Jay A. Gregory, MD

What's It All About?

"What we are seeing is the doctor simply becoming a cog—a unit in an enterprise that is basically a business," said Arnold S. Relman, MD, editor-in-chief emeritus of the *New England Journal of Medicine*. Figuratively speaking, *cog* means a person or thing having a minor but necessary function in the running of any system or organization. I'm sorry, Dr. Relman, but I haven't spent the past 20 years of my life merely to become a cog in some corporate web.

"You're here, you've made it. All those years of hard work have paid off," Gordon Deckert, MD, said to the incoming class of first-year medical students in late August 1972. Years of hard work for what? Work that now allows me to generate more than $1 million in revenues for my hospital each year. Work that allows an internist to generate approximately $800,000 for his or her hospital yearly. Work that allows the family practitioner to generate approximately $500,000 for his or her hospital yearly.

No, this work has led us to solemnly pledge ourselves to consecrate our lives to the service of humanity; to give our teachers the respect and gratitude which is their due; to practice our profession with conscience and dignity; to keep the health of our patients as our first consideration; to respect the secrets which are confided in us; to maintain by all means in our power the honor and the noble traditions of the medical profession; to remember that our colleagues will be our brothers and sisters; to not permit considerations of religion, nationality, race, party politics, or social standing to intervene between our duty and our patients; to maintain the utmost respect for human life from the time of conception. Even under threat we will not use our medical knowledge contrary to the laws of humanity. We all made the promises solemnly, freely, and upon our honor.

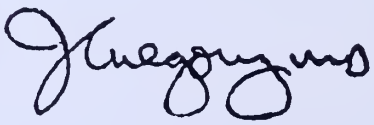
So what has happened to our honor? Has it been replaced by greedy corporate America and the thrust for the almighty dollar? I don't think so! Nowhere could I find in any translation of the Hippocratic Oath, written during the 4th century BC, that we should place the dollar above all else. So why rush to become "vertically integrated" with individuals or entities that exist solely to bring a profit to the bottom line? Are the managed care blues driving us into corporate arms? Why would Dr. Jason Rosenbluth of Volpe, Welty, & Co., a San Francisco investment-banking firm, predict that by the year 2000, 56% of all physicians will be in groups, up from 32.6% in 1991? Why are the Blues, Aetna, Cigna, Travelers, and the Prudential buying up physicians' practices all across our country? Why are you considering selling your practice to these entities or even to the hospital at which you practice? Haven't you heard that hospital administrators and physicians make strange bedfellows?

If you are truly concerned about your future, then do something positive which will keep you in control of your patients and therefore your practice of medicine. Consider joining the Oklahoma Physician Network (OPN), your new independent practice association (IPA), and your OSMA-endorsed managed care organization (MCO). Your support and participation in these two entities, entities which you will own and control, will ensure that you make all decisions concerning the delivery of health care to your patients, as well as all the business decisions concerning these companies.

The grand design of all the giant health cartels is to divide and exploit Oklahoma physicians. Perhaps the poet W.H. Auden had such relationships in mind when he said, some thirty years ago, "We are here on earth to help others, but what the others are here for, I cannot say."

Pay careful attention to these pages in the months ahead to learn more about the Oklahoma Physician Network and your MCO. Also please be responsive to mailings that you will be receiving from your OSMA on a whole host of issues that will be of grave concern to you and your fellow physicians.

Remember, your colleagues are your brothers and sisters, and the health of your patients shall be your first consideration.



Parental and Medical Over-Administration of Acetaminophen Causing Lethal Hepatotoxicity in a 10-Year-Old

Abdul Nadir, MD; Robert McFadden, MD; John Griggs, MD; Harlan I. Wright, MD; Stefano Fagiuoli, MD; David H. Van Thiel, MD

A 10-year-old girl with acute influenza B virus disease was given repetitive doses of acetaminophen by her mother to reduce the child's fever. When finally seen by trained medical personnel, the child was experiencing abdominal pain, nausea, and vomiting, which are classic signs of acetaminophen hepatotoxicity. Despite these findings, the child was given additional acetaminophen and experienced lethal hepatotoxicity.

ACETAMINOPHEN IS A COMMONLY USED ANTIPYRETIC analgesic agent that is readily available over the counter. Despite its universal availability, its excessive and uncontrolled use is associated with hepatotoxicity. Acetaminophen hepatotoxicity can cause serious acute illness and in some cases is fatal.[1] Fatal outcomes usually are a result of attempted suicide with acetaminophen or the use of excessive amounts of acetaminophen by an individual with upregulated hepatic P-450 drug metabolizing enzyme systems, which are responsible for acetaminophen metabolism, as a consequence of chronic use of either ethanol or an anticonvulsant.[2,3] Although acetaminophen is a very common cause of drug toxicity in children under ten years of age, mortality occurring as a consequence of acetaminophen use is an exceedingly uncommon event.

Herein is reported a case of acetaminophen-induced mortality occurring in a 10-year-old girl.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112.

Case Report

On January 21, 1993, a 10-year-old girl was referred from a community hospital to a primary care center in Oklahoma City for markedly elevated hepatic enzyme levels and an altered mental status. Twelve days earlier, she had experienced a flu-like syndrome consisting of a sore throat and muscle aches. She was treated by her mother with repetitive doses of acetaminophen plus an additional non-aspirin-containing over-the-counter preparation (Alka-Seltzer). Four to five days later, the patient complained of fatigue and anorexia. She was seen in an out-patient clinic and was given additional orally administered acetaminophen consisting of two extra-strength tablets. Her symptoms were exacerbated and she additionally developed nausea, vomiting, and generalized abdominal pain. On January 19, 1993, she was taken to an emergency room where a diagnosis of a viral syndrome was made and she was admitted to the hospital for observation. On January 20, 1993, a serum acetaminophen level obtained two days after her last dose of acetaminophen was reported as being 49 (normal ≥50 μg/ml). Because of the severity of the hepatic biochemical findings occurring in association with a decline in her mental status, she was transferred to the primary care center.

At the center, she was found to demonstrate decorticate posturing, have a pulse of 110 beats per minute, a respiratory rate of 36 per minute, and a blood pressure of 135/70 mmHg. She was afebrile. Scant crusted blood was evident in her mouth. Keyser Flescher rings were not present. She had no palpable hepatosplen-

omegaly. The clinical laboratory findings obtained at the center are summarized in Table 1. Hepatitis A, B, and C serologies, a monospot test, and various CMV titers were all negative. An autoimmune panel consisting of an ANA, RF, SM antibodies, SSB (LA) and SSA (RO) were all negative. Her alpha-1 antitrypsin level was 109 mg/dl (n193-224) and her ceruloplasmin level was 21 mg/dl (n121-35). Her serum copper level was 80 μg/dl (n170-150). A CT scan of the abdomen demonstrated a liver volume of 958 cc and a normal spleen size. A CT of the head was unremarkable.

Table 1. Clinical Laboratory Data Available as the Greatest Reported

	January 19, 1993	January 21, 1993
White blood cells	2.1 (4.8-10.8 cells x 10^3)	10.7 (4.5-13 cells/mm^3)
Hemoglobin	14.5 (12-16 g/dl)	11.2 (11.5-14.5 g/dl)
Hematocrit	38.5 (33-47%)	32.3 (31-46%)
Platelets	174 (150-400 cells x 10^3/mm^3)	136 (150-433 cells/mm^3)
AST	2,364 (7-34 μ/1)	14,150 (0-30 μ/1)
ALT	1,402 (4-37 μ/1)	8,690 (0-40 μ/1)
Alkaline phosphatase	204 (30-103 μ/1)	448 (70-365 μ/1)
Bilirubin (Total)	0.6 (0.4-1.5 mg/dl)	2.9 (0.0-0.4 mg/dl)
BUN	8.6 (7-19 mg/dl)	23 (5-25 mg/dl)
Creatinine	0.5 (0.7-1.4 mg/dl)	3.4 (0.5-1.5 mg/dl)
Albumin	4 (3.7-5.2 g/dl)	3.3 (3.5-5.2 g/dl)
Acetaminophen	49 (0-50 μg/ml)	less than 10 μg/ml)
PT	normal	63 seconds
PTT	normal	55 seconds

Because of her respiratory distress, the patient was intubated and placed on mechanical ventilation. Within 24 hours of admission she developed acute renal failure. An ultrasound study of the kidneys demonstrated an increased echogenicity of both kidneys. Renal dialysis was instituted. The patient experienced a grand mal seizure. A CT scan of the head demonstrated diffuse cerebral edema. The patient rapidly deteriorated and was pronounced dead on January 22, 1993.

An autopsy revealed a bland severe centrilobular necrosis of the liver involving 50 to 70% of hepatic lobules. The kidneys demonstrated renal tubular epithelial necrosis. Generalized benign lymph adenopathy was present. Foci of intrapulmonary hemorrhage were present and were cultured at autopsy. Subsequently, these were found to be positive for influenza B virus.

Discussion

Acetaminophen is metabolized largely in the liver by conjugation with glucuronic acid or sulphate. The metabolites formed are then excreted by the kidneys.[4] The cytochrome P-450 enzymes of the mixed function oxidase systems of the liver and, to much lesser extent, the kidney, catalyze the oxidation of acetaminophen to N-acetyl-p-benzoquinonimine (NABQI), a highly reactive metabolic intermediate.[5,6] The increased formation of this reactive intermediate (NABQI) as a consequence of P-450 induction is responsible for the hepatic necrosis caused by acetaminophen. Normally, NABQI is detoxified endogenously by conjugation with glutathione. Under circumstances of increased production, as occurs after an acetaminophen overdose and when acetaminophen is taken after a relatively prolonged fast as occurs with many acute illnesses such as influenza B, glutathione stores are depleted and the NABQI formed reacts irreversibly with cellular macromolecules, resulting in hepatic injury that can progress to hepatic failure.[7,8]

On the basis of published literature, it appears that acute nephrotoxicity induced by acetaminophen occurs less often than does acute hepatotoxicity.[9] Nonetheless, acute renal failure occurring after acetaminophen overdosage has been reported to occur both with and without coexistent hepatic injury.[10,11]

The hepatic lesion induced by acetaminophen is characterized by a bland centrilobular necrosis.[12] The renal lesion associated with acetaminophen injury consists of tubular necrosis with fatty change early and renal calcification later.[13] Rarely, myocardial necrosis has been reported to occur in cases of acetaminophen poisoning.[14] Hepatic toxicity due to acetaminophen ingestion can occur with single doses of more than 7.5 grams in adults or 150 mg per kg of body weight in children.[7] The actual toxic dose in most cases is usually considerably greater than these cutoff values.[8]

Poisoning as a consequence of uncontrolled acetaminophen overdosage has increased recently in adolescents. In a 12-year study, Hawton et al reported the frequency of acetaminophen poisoning in adolescents has increased from 23.4% to 48.3% of adolescent attempted suicides.[15] Despite this apparent increase in acetaminophen toxicity in older children, only a very few cases of mortality occurring as a re-

sult of acetaminophen toxicity in children 10 years or less in age have been reported.[1,16-18] These consist of seven cases of children between the ages of 13 and 72 months. Four of these seven children were reported to have chronic acetaminophen poisoning, whereas only three had a history of acute acetaminophen overdosage.[1]

The reported low incidence of severe acetaminophen toxicity in children after ingestion of large doses of acetaminophen may be related to the greater capacity of the liver of children to metabolize acetaminophen via non-toxic pathways. Moreover, the relatively greater sulphate and glutathione stores of children also may contribute to reducing the toxicity of acetaminophen in children.[19]

In summary, this is a report of a case of accidental (iatrogenic) acetaminophen-induced mortality occurring in a child in Oklahoma. Despite the presence of abdominal pain and vomiting, which are classical signs of acetaminophen hepatotoxicity, the diagnosis was not considered and additional acetaminophen was prescribed when the child was seen by medical specialists. This case provides a clear message that children with viral syndromes who are given acetaminophen and subsequently develop nausea and vomiting should be investigated for possible hepatotoxicity. If hepatic injury is identified, these children should be treated aggressively with oral acetyl cystine to either supplement or restore their hepatic glutathione stores. Evidence of progressive hepatic and/or renal injury should initiate referral to a liver transplant center.

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The Authors

David H. Van Thiel, MD, is medical director of transplantation at the Oklahoma Transplantation Institution (OTI) and director of the Hepatic and Digestive Disease Center (HDDC) at Baptist Medical Center of Oklahoma (BMCO) in Oklahoma City.

Drs. Nadir, McFadden, Griggs, Wright, and Fagiuoli are on staff at OTI.

Anticardiolipin Antibodies: A Case Report

Neil B. Kimerer, MD; Michael A. Tribbey, MD; Phillip J. Wright, MD; E. Edward Beckham , PhD; Lynn L. Myers, MD

The authors describe a relatively uncommon case of primary anticardiolipin syndrome appearing in a patient whose symptoms are manifested predominantly in the central nervous system.

ON NOVEMBER 8, 1993, THE WIFE OF A 55-YEAR-old man brought him to the emergency room with moderate to severe gastrointestinal bleeding. A thorough inpatient GI workup failed to reveal significant pathology. His mentation was very erratic, and his wife informed the gastroenterologist that he had been mistreating her for the previous three years. His mental functioning had so deteriorated that he had been unable to work for the past three years. Three years prior he had been admitted to a state mental hospital but they were unable to diagnose him. He left having had minor neuroleptics prescribed, which had not helped. No physical impairment nor abnormal laboratory results were found.

The patient was oriented, with poorly organized thought processes. He was hallucinating that people were in the house and was paranoid about his wife meeting people in the house and going to bed with them while he watched. He thought he was invisible to them. His manner and attitude had made major changes about two years prior to the present hospitalization, and he had become seclusive. His activities had become limited, and he took little interest in what was going on around him. His paranoia about his wife had increased. She thought he had had a stroke but had not taken him to a physician. The patient was not aware that his behavior and thinking had changed. He had been awarded disability at that time and his wife continued working, leaving him alone at the house all day.

The patient was transferred to a senior diagnostic center after the GI workup had been completed. His right pupil was 1.0 to 1.5 mm larger than the left. He was noted to have bilateral spasticity of both lower extremities with unsustained clonus, bilateral extensor plantar response, and an absence of cerebellar abnormalities. His EEG was very disorganized, with generalized slowing noted. No periodic complexes suggestive of viral or prior infections were noted.

MRI showed periventricular white matter changes without infarcts or atrophy. Laboratory work-ups demonstrated the following: Westergren ESR 127, 129, Wintrobe ESR 59. Vital signs normal. Normal fasting chem 20, CBC, thyroid profile, Mg level, Lyme disease (Borrelia) titer 0.44; CEA 2.4; CH 100, 73 (Normal 40-80 µmL) ASO titer negative, Complement C-3 105,(85-193 Mg/DL) and Complement C-4 15 (12-36 Mg/DL) ECG were normal. Chest x-ray showed some resolving cloudiness compared with admission films.

Neurology consult was obtained (MAT). Further laboratory studies revealed IgG antibodies 2540, (normal <1349), IgA 847, (normal <312) ANA titer 1:80 , UA negative, Spg. 1.010, Ph 5.0; Anti-DNA (ELISA) 29 IU/ml (<30 = Negative). Nonreactive HIV-1 AB. Spinal tap was completely normal for cell count, pres-

Direct correspondence to Neil B. Kimerer, MD, 2600 Northwest Expressway, Oklahoma City, OK 73112.

sure, proteins, and glucose, and negative for AFB, fungus India ink prep and VDRL. There were some atypical bilobed mononuclear cells. Bacterial and fungal culture were negative. Serum protein electrophoresis showed beta globulin–containing beta lipoprotein, C3 complement, and transferrin with diffuse elevation of gamma globulin, suggesting a possible polyclonal gammopathy.

Iliac bone marrow biopsy demonstrated mild nonspecific lymphocytosis with focally increased areas of cellularity, but all elements were present in appropriate degrees of maturation. There was no indication of medullary infiltration with red cell series normoblastic, and the iron stain failed to reveal demonstrable material.

Psychological testing battery revealed a WAIS-R Verbal IQ of 90, performance IQ of 76, and a full-scale IQ of 84 (EEB). The patient was a college graduate from a major university at age 22. He obviously had lost intellectual capacity. The tests revealed predominant organic disturbance rather than a functional psychosis or affective dysfunction.

Rheumatology consult was obtained (PJW). Further laboratory studies revealed a negative rheumatoid factor, IgG antibodies 2240, (normal <1349); IgA antibodies, 730 (normal <312); and IgM 199 (normal <352); anticardiolipin IgM antibodies 350 (normal <10).

The patient's behavior was grossly out of contact with reality. He liked to "streak," in which he disrobed and wandered around the unit. He slept well, ate well, and was easily distracted from his streaking, but he was unpredictable and not reliable in going to the activities which were ordered for him. Fortunately, he was not belligerent or combative and was easily managed. His cognitive functions seemed to be intact, and he was coherent even though his speech was slurred.

His medications were lorazepam 1 mg q 12 hrs, pimozide 1 mg q 12 hrs, thiothixene 2 mg hs and 5 mg each morning, and alternagel 300 ml three times daily. He was also given a short course of prednisone 20 mg BID for a week and then tapered during the last week of stay and was discharged on no steroid.

Discussion

The autoimmune concept of disease has required some six decades to develop beginning with Libman (sic) and Sachs[1] and expanded by Aggeler[1] et al in 1946, with very few papers between. The cardiolipins are classed with other phospholipids and are known to be located in the inner cell membrane of mitochondria . Other phospholipids are found in endothelial cell membranes, platelets, and brain tissue.[3] With the advent of ELISA (enzyme linked immunosorbent assay), the detection of antiphospholipid antibodies has been greatly enhanced, and many diverse diseases have been shown to share these common factors.[1,3-13] Systemic lupus erythematosus (SLE),[5,11,14-16] repeated spontaneous abortions,[18] in which they seem to be the most frequent, endocarditis,[11,18,19] retinal vascular disease,[19] rheumatic disorders,[18] and several neurologic disorders,[11,12,17,18,20-22] thyroiditis, venous thrombosis,[18,19,21,23] thrombocytopenia,[23] and major vasculitis,[18,19,21,23] as well as other altered physiological processes.

SLE has been studied the most extensively, but the details have yet to be identified and Mackworth-Young[24-27] has proposed that there is a condition of a primary antiphospholipid syndrome as a separate entity. Diseases with and without the lupus anticoagulant (LA)[4,6,7,20,23,28,29] are known to occur, and it is postulated that the cardiolipin antibody (AB) may react with phospholipids. Hughes et al have postulated that this may occur within the spectrum of SLE syndrome as well as in other diseases, or separately.[14,24-26]

Feinstein[23] proposed the term *lupus anticoagulant inhibitor,* which is now utilized in separating the LA from that which is not SLE. Our patient did have the inhibitor, so he is free of the SLE manifestations at this time. Whether or not that will change remains to be seen. A routine test for these factors is the activated partial thromboplastin time (APTT), which is one indication of the activity of the inhibitor. The patient therefore falls into the category of a primary anticardiolipin syndrome. Levine and Welch[11] identified fewer than 70 such cases in the world literature in 1987.

Our patient had his primary symptoms in the central nervous system (CNS), and there are relatively few references to that organ system in the literature.[6-8,10-13,17,20-22,27,30] We have focused the references to this organ system, and there are multiple overlaps in the symptomatology that can be presented. Our patient showed positive peripheral neurological signs and functional organic brain disease but, surprisingly, no definitive infarcts nor occlusions large enough to be seen on the MRI even though they did affect the EEG.

This disorder is one of many autoimmune antiphospholipid disorders, all of which have very vague distinguishing features to differentiate them from one another. Often, from the literature, the symptoms and chemical parameters shift from one to another, so the

distinction becomes even more vague. SLE also seems to be closely associated with all of the various types. The differences in the diagnoses seem to depend on which organ system reflects the primary site of activity at that particular time. From time to time any single patient may be accurately diagnosed with different names, valid only for that particular time. In our patient, serological tests for syphilis were negative, became positive, and reverted to negative within the span of a few days. It may be that this entire galaxy of disorders is a single disease with multiple variations.

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The Authors

Neil B. Kimerer, MD, is certified in psychiatry and is clinical professor emeritus in the Department of Psychiatry and Behavioral Sciences at the University of Oklahoma Health Sciences Center (OUHSC) in Oklahoma City.

Michael A. Tribbey, MD, is a board certified neurologist with a private practice in Oklahoma City.

Phillip J. Wright, MD, is an internist with special competence in rheumatology and associate professor of clinical medicine in the OUHSC Department of Medicine.

E. Edward Beckham, PhD, is a diplomate in clinical psychology (ABPP) and clinical associate professor in the OUHSC Department of Psychiatry and Behavioral Sciences. He has a private practice in clinical psychology.

Lynn L. Myers, MD, is certified in clinical and anatomic pathology and is director of the Department of Pathology at Deaconess Hospital in Oklahoma City.

Campylobacter fetus Presenting As a Septic Pleural Effusion: A Case Report

Abdul Nadir, MD; Harlan I. Wright, MD; Farah Nadir, MD; D.H. Van Thiel, MD

The authors describe a rare case of *Campylobacter fetus* occurring in a patient who showed no overt evidence of immune incompetence. The patient presented with severe cellulitis of the legs, a chronic low grade fever, and a history of recent exposure to sick calves.

CAMPYLOBACTER FETUS IS A SHORT, COMMA shaped, motile, microaerophilic, gram-negative bacillus.[1] It is a global zoonosis and an established cause of infectious abortion in cattle and sheep.[1] Vinzent et al, in 1947, described the first case of *Campylobacter fetus* subspecies *fetus* as a cause of infection in humans.[2] Since then, it has emerged as an unusual pathogen in human beings.[3-15] In contrast to *Campylobacter jejuni,* a closely related organism, *C. fetus* rarely is a cause of gastroenteritis.[3,16] Instead, it usually appears in humans as a systemic infection such as endocarditis,[17-19] meningitis,[7-9,20] aortic aneurysm,[21] thrombophlebitis,[22] septic arthritis,[23] or pelvic inflammatory disease.[24] It is the most common etiologic agent in systemic campylobacteriosis.[15,25,26] Pulmonary involvement with this organism is distinctly unusual.[5,10] Herein, we report a case of *C. fetus* septic pleural effusion and a severe cellulitis of the legs occurring in a patient with chronic liver disease.

Case Report

A 62-year-old male farmer presented for an evaluation of possible liver transplantation. He had a three-year diagnosis of post-necrotic cirrhosis due to hepatitis C infection, presumably acquired as a result of a blood transfusion.

When first seen on July 19, 1993, the patient complained of exertional shortness of breath, intermittent chills, night sweats, and a dry cough. He denied all gastrointestinal symptoms and reported having had low-grade fevers and night sweats for six months. He had been intermittently treated for these symptoms with cefuroxime 250 mg two times a day with minimal relief. Two weeks prior to this clinic visit, he was treated with oral cefuroxime for a left arm cellulitis. He admitted to having had one episode of pneumonia and two episodes of presumed spontaneous bacterial peritonitis during the year prior to his present illness. In addition to cirrhosis, his medical history was positive for a benign colonic polyp, kidney stones, hypothyroidism, and gout, and he had had a hemorrhoidectomy and vasectomy. He had no history of alcohol, drug, or tobacco abuse.

As part of his profession, he has close contact with cattle. In the first week of July, two of his calves were sick with diarrhea and pneumonia, and one died.

On physical examination, he appeared chronically ill with a BP of 133/73 mmHg, pulse of 87/minute, respiratory rate of 20-24/minute, and an oral temperature of 98.3° F. He had multiple depigmented scars on his upper and lower extremities, and there was no lymphadenopathy. A few spider angiomata were noted on his thorax, and he had bilateral gynecomastia. Reduced breath sounds and dullness were noted over

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112.

his right hemithorax extending inferiorly from the fourth intercostal space. He had a moderate amount of ascites and a flapping tremor. The remainder of his physical examination was unremarkable.

His white blood cell count was 9.6 (N=4-10.4 cells/mm^3) with a differential count of 34% bands, 51% polys, 11% lymphs, and 3% monos. His platelet count was 85,000 (N=150-433 cells/mm^3). His prothrombin time was 16 seconds (N=10-14 seconds). A chest x-ray demonstrated a moderately large right pleural effusion which layered out on decubitus films. The admission and laboratory data were otherwise unremarkable. Thoracentesis removed six liters of straw-colored clear pleural fluid. The pleural fluid was a transudate containing 495 WBC/mm^3 with a differential count of 34% polys and 59% lymphocytes.

On July 20, the patient spiked a fever to 102°F and complained of leg pain. Clinical examination revealed a new onset bilateral lower leg cellulitis. Blood cultures were obtained and the patient was started on intravenous cefazolin 1 gm every 8 hours. A Gram stain of his sputum revealed many white cells with gram-positive rods and cocci.

An intermediate PPD test was negative, and skin tests for candida and trichophyton were positive. The pulmonary function tests revealed a pattern consistent with severe restrictive lung disease. A full colonoscopy and upper gastrointestinal endoscopy revealed portal hypertensive gastropathy and enteropathy, and also esophageal varices. The signs of an infectious enteritis were not found. At this time, the patient continued to have fever spikes despite intravenous cefazolin.

On July 25, his blood and pleural fluid cultures from July 20 were reported as positive for gram-negative bacilli. The gram-negative bacteria were later identified as *Campylobacter fetus* subspecies *fetus* by the Oklahoma state microbiology laboratory. These cultures were incubated at 25°, 36°, and 42°C in an atmosphere containing 10% carbon dioxide, 5% oxygen, and 85% nitrogen. No growth occurred at 36°C, while strong growth was observed at 42°C and weak growth was noted at 25°C. The organism isolated was a curved rod with bipolar flagella, and was motile and positive for oxidase but negative for hippurate. It was minimally sensitive to cephalothins and resistant to nalidixic acid.

The patient was started on intravenous imipenem (1 gm every six hours) and ciprofloxacin (400 mg every 12 hours). His fevers and lower leg cellulitis abated in 48 hours, and he was discharged on July 31. At follow-up during the second week of August, a minimal right-sided pleural fluid was seen on the chest x-ray, and a repeat diagnostic thoracentesis was performed. The pleural fluid was clear, straw-colored, and culture negative. Currently, the patient is doing well, with no clinical or radiologic evidence for reaccumulation of pleural effusion.

Discussion

C. fetus is distinguished from other species of campylobacter (*jejuni* and *coli*) on the basis of its growth characteristics, antibiotic sensitivity, and clinical features.[1] *C. fetus* usually grows at 25°C but not at 36°C. A few strains of *C. fetus,* however, can grow at both 25°C and 42°C.[27] In vitro, *C. fetus* is moderately susceptible to cephalothin but is resistant to nalidixic acid.[1] In contrast, *C. jejuni* and *C. coli* grow at 36°C and 42°C and are sensitive to nalidixic acid but resistant to cephalothin.[1]

In humans, *C. fetus* infections typically are manifested as a subacute low grade fever with bacteremia.[15,27] Elderly and debilitated patients appear to have an increased rate of *C. fetus* infections, particularly when they have an associated condition such as alcoholism, immune deficiency, or surgical or medical stress.[6,15,26]

Clinically, *C. fetus* infections present as either a bacteremia, endocarditis, vasculitis, or cellulitis. In contrast, *C. jejuni* infections are well described in humans and occur almost exclusively as an enteric infection with diarrhea. The range of signs and symptoms of *C. jejuni* infection runs from asymptomatic colonization with *C. jejuni* to a severe ileocolitis which typically occurs in infants and children.

In contrast to the widely known pathogenesis and epidemiology of *C. jejuni,* little information concerning *C. fetus* infection is known. The acquisition of *C. fetus* infection is presumed to occur via the oral route. Because enteritis is rare with *C. fetus,* it is rarely found in stool.[26]

Pulmonary involvement with *C. fetus* is an extremely unusual manifestation. A review of English language literature revealed only three prior cases of *C. fetus*–associated pulmonary infections.[5,10] Targan et al[5] reported a case of a 24-year-old intravenous drug abuser who developed aspiration pneumonia after an overdose of diazepam and ethchlorvynol. *C. fetus* was isolated in mixed culture with two other organisms from the patient's empyema fluid, while the blood cultures were negative for *C. fetus.* The patient was treated successfully with a four-week course of intravenous gentamicin.

Hoover et al[10] reported a case of a 64-year-old male

with stage two squamous cell carcinoma who underwent a pneumonectomy. Two weeks postoperatively, he developed a febrile illness with a pleural effusion and both the pleural fluid and blood cultures grew out *C. fetus*. Their patient responded to intravenous antibiotics. Interestingly, their patient reported having sick dogs with diarrhea immediately prior to his hospitalization.

In a third case report, a patient on cyclophosphamide therapy for lymphosarcoma developed a *C. fetus*–positive pleural infection occurring in association with a bacteremia.[5]

In contrast to these three reports, the present case is unusual in that it is the first case of *C. fetus* pleural effusion occurring in a patient without overt evidence of immune incompetence. The three prior cases had histories of intravenous drug abuse or neoplasia. The present patient did have cirrhosis, which is associated with a variety of immunologic alterations, but he was believed to be immune competent because of positive skin tests for candida and trichophyton. This case is unusual also because it presented with a cellulitis and chronic low grade fever. There was no history of operative trauma to the lung or pulmonary aspiration. Finally, this case demonstrates that *C. fetus* infection can occur in an immunocompetent host as a chronic smoldering infection. Although it cannot be proven in the present case, it is likely that the prior pulmonary infection in this patient was the primary manifestation of his *C. fetus* infection that slowly resolved, leaving an infected pleural fluid as a nidus for subsequent intermittent bacteremia that caused the lower leg cellulitis.

Retrospectively, the factors that should have pointed to a diagnosis of *C. fetus* infection in this man included his recent history of working with sick calves, the chronicity of the infection as manifested by his history of intermittent fever, and the finding of a cellulitis of the legs in the absence of any recent history of leg injury. Although no cultures were obtained from either of the sick calves, the veterinarian who treated them and did the necropsy on the dead calf reported to the present investigator that the animals probably had a *C. fetus* infection that could have been transmitted to the patient herein reported.

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The Authors

David H. Van Thiel, MD, is medical director of the Oklahoma Transplantation Institute (OTI) and director of the Hepatology and Digestive Disorder Center (HDDC) at Baptist Medical Center of Oklahoma (BMCO) in Oklahoma City.

Drs. Abdul Nadir, Harlan I. Wright, and Farah Nadir are on staff at OTI.

SPECIAL

Hunting-Related Spinal Cord Injuries Among Oklahoma Residents

Cheryl Price, MPH; Sue Mallonee, RN, MPH

While major causes of traumatic spinal cord injuries have been described as motor vehicle crashes, falls, sports-related events, and injuries related to violence, causes of serious consequence but of lesser magnitude have not been widely reported. This report, drawn from Oklahoma State Department of Health spinal cord injury surveillance data, describes the incidence and circumstances surrounding hunting-related spinal cord injuries. All of the injuries resulted from falls from trees or tree stands. The incidence rate of injury was less than one per 100,000 licensed hunters. Half of the injuries resulted in neurological damage severe enough to result in permanent paralysis or death. The circumstances of the injuries suggest a need for educating hunters concerning safe hunting practices and safe tree stand use, including inspection of trees for weak or dead branches, and use of a safety belt when ascending, descending, or sitting in a tree or tree stand.

TRAUMATIC SPINAL CORD INJURIES CAN BE AMONG the most debilitating of all injuries, often leaving survivors with neurological deficits ranging from loss of sensation to permanent paralysis. Major causes of these debilitating injuries have been widely reported as motor vehicle crashes, falls, sports-related events, and injuries resulting from violence.[1-3] Less well known, however, are causes of serious consequence but of lesser magnitude, which differ by region. In Oklahoma, hunting is a popular pastime, with approximately eleven percent of the population holding a hunting license.[4] When reporting of traumatic spinal cord injuries began in 1987, hunting-related activities began to become apparent as a unique cause of spinal cord injury. This report will estimate the incidence of hunting-related spinal cord injuries occurring among Oklahoma residents and describe the circumstances surrounding the injuries.

Methods

Data from the surveillance system of the Injury Prevention Service of the Oklahoma State Department of Health were examined for cases occurring in the five and one-half year period from October, 1987 through March, 1993.

Statewide traumatic spinal cord injury reporting to the Injury Prevention Service utilizes a multi-level system of reporting. Physicians and hospitals report acute injury, rehabilitation centers report post-acute injury, and the State Medical Examiner reports fatal injury. In addition, hospital medical record departments began reporting acute injuries based upon discharge diagnosis codes in 1992. Only Oklahoma residents are included in the surveillance system. The surveillance case definition of a traumatic spinal cord injury is any acute, traumatic damage to the spinal cord which results in any degree of sensory or motor deficit; the deficit can be temporary or permanent. Injury to the nerve roots or spinal plexus is not included. Among injuries which are fatal before hospitalization, only cases with a singular cause of death from spinal injury or clearly having a severed spinal cord are included in the surveillance database.

Direct correspondence to Cheryl Price, MPH, Injury Prevention Service–0307, Oklahoma State Department of Health, 1000 Northeast Tenth Street, Oklahoma City, OK 73117-1299.

A hunting-related spinal cord injury was defined for this study as a spinal cord injury occurring while the individual was participating in any form of hunting or during preparation for hunting. Denominator data for numbers of hunting licenses sold were obtained from the Oklahoma Department of Wildlife Conservation[5] and averaged for the years 1987 through 1992.

Results

Ten hunting-related spinal cord injuries were reported during the study period; the number of injuries ranged from one to three per year. All injured persons were hospitalized; no fatalities occurring at the scene of the injury were identified. This resulted in an annual rate of less than one (0.5) spinal cord injury per 100,000 hunting licenses sold.

Each of the ten injuries was caused by falling from a tree while hunting or during preparation for hunting; none was caused by firearm injury. Seven were reported to be in a tree stand and one was in a deer blind, while no detail regarding use of a tree stand was available for the remaining two. The injured fell from estimated heights of 15 to 30 feet. Specific circumstances surrounding the falls and the number of known applicable cases included: fell when tree limb broke (3), fell while building or setting up a tree stand (2), fell while descending from a tree (1), fell after drinking and falling asleep in the tree (1), and fell as a result of structural failure of a one year-old homemade stand (1). One person was bow-hunting from a tree stand. Three persons reported landing on their head or upper back and three reported landing on their buttocks. Darkness was reported to have contributed to one of the injuries.

The injured were all white males evenly dispersed in age from 18 to 57 years of age (Table 1); the average age was 41 years. Eight were injured at the thoracic or lumbar spine level, resulting in paraplegia/paresis, and two were injured at the cervical spine level, resulting in quadriplegia/paresis (Table 1). Half of the injuries resulted in severe neurological damage, with four persons incurring complete injuries resulting in permanent paralysis and one incomplete quadriparetic injury resulting in a functional level of sensation only (no motor function remaining). One of the persons with a complete injury died shortly after admission to a nursing home, resulting in an overall case fatality rate of 10%.

The initial acute care hospitalization period ranged from 4 to 73 days; the average was 26 days. Half the patients were hospitalized two weeks or less (Table 1). Four were discharged to rehabilitation centers, one person too severely injured for rehabilitation was discharged to a nursing home and subsequently died, and at least one other person received outpatient therapy after being discharged home. Among the four persons admitted to rehabilitation centers, stays ranged from 48 to 131 days and the average was 78 days.

The injuries were not distributed equally by month or day or time of injury, or by geographical location of the injury. All but one injury occurred in October or November, coinciding with Oklahoma deer hunting seasons. The one March injury occurred as the patient was building a tree stand near his home. Seven of the injuries occurred on Friday, Saturday, or Sunday, and eight of the injuries occurred between 5:00 AM and noon. Three of the injured persons reportedly remained exposed on the ground for 2 to 5 hours after the injury before being found by searchers. All but one of the injuries occurred in two three-county areas (Fig. 1). Half of the injuries occurred in the injured person's home county and three others occurred in counties bordering the home county.

Table 1. Selected Characteristics of Hunting-Related Spinal Cord Injuries, Oklahoma, October 1987 Through March 1993

Characteristic	Number (%)
Age	
15-24	2 (20)
25-34	2(20)
35-44	2 (20)
45-54	2 (20)
55-64	2(20)
Type and Degree of Injury	
Incomplete paraparesis	5 (50)
Incomplete quadriparesis	1 (10)
Complete paraplegia	3 (30)
Complete quadriplegia	1 (10)
Length of Acute Hospitalization	
Less than 2 weeks	5 (50)
2 to 4 weeks	2 (20)
More than 4 weeks	3 (30)

Discussion

To our knowledge there were no other published studies specifically addressing hunting-related spinal cord injuries in the literature. There were, however, several reports addressing hunting injuries in general, or more commonly, addressing firearm-related hunting injuries.[6-11] A study focusing on tree stand-related injuries among deer hunters in Georgia, drawn from Georgia's Uniform Hunter Casualty Reports for a ten-year period, found 594 deer hunting-related injuries, 214 of which were tree

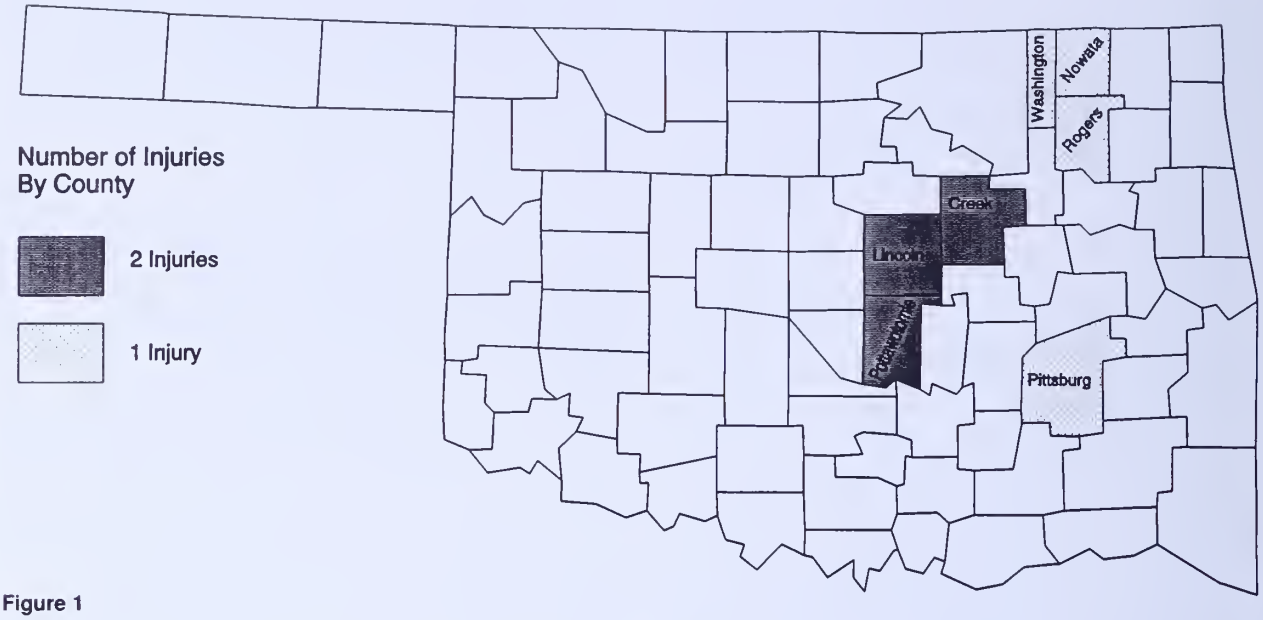

Figure 1

stand-related and 16 of these were cervical spine fractures.[6] Similarly, in 1991, a study from one hospital in Georgia found 19 tree stand–related fall injuries in a period of approximately seven years.[7] An evaluation of emergency department patients at a Wisconsin hospital during two hunting seasons revealed 104 hunting-related injuries, with eight patients incurring vertebral body fracture or significant back strain; 17 of the injuries resulted from falls and twelve of these were specific to tree stands.[8] A Swedish study of firearm-related hunting fatalities reported two fatalities occurring when firearms fired after unintentionally being dropped from hunting towers.[9] Finally, two studies describing hunting-related firearm injuries did not address spinal cord or tree stand–related injuries.[10,11]

Statistics for overall hunting-related injuries in Oklahoma are not available for comparison for the years of our study. The Oklahoma Department of Wildlife Conservation, however, has collected data on *firearm-related* hunting injuries for over 25 years (data includes bow-and-arrow and other projectile apparatus). From 1987 through 1992, they received reports of 86 firearm-related hunting injuries, resulting in a firearm hunting injury rate of 4.1 per 100,000 hunting licenses sold.[5] Fifteen of the injuries were fatalities, resulting in a firearm hunting injury case fatality rate of 17%.

Tree stands were known to be used by the majority of injured in this analysis and are commonly used while hunting deer in Oklahoma. A survey of a stratified random sample of 2,027 hunting license holders in 1991-1992 by the Oklahoma Department of Wildlife Conservation revealed that 45% (638/1408) of the individuals who hunt deer reported hunting deer from an elevated stand (S. DeMaso, personal communication). Tree stands are popular because of the advantages they afford the hunter, including an expanded field of vision and leaving minimal ground scent. While tree stands may be either purchased or constructed by the user, little information on the type of stand used by the injured in this report was available. However, one person was known to be constructing a stand, another was descending while in a deer blind, another fell when he stepped onto the stand and a lock or chain did not hold, and finally, one man fell because nails had loosened on the wooden stand he had constructed the prior year.

The uneven geographic dispersion of the injuries is difficult to evaluate, especially in light of the small number of cases. Data on overall hunting injuries not limited to spinal cord injuries would likely be helpful in exploring the relevance of this finding; however, the clusters of injuries may be related to topography and/or locations of populations of hunters and game.

While the number of hunting-related spinal cord injuries was small, the steady occurrence and seriousness of the injuries, as well as the great potential for prevention, suggest further attention should be directed to the problem. Health practitioners and

hunting safety instructors should educate hunters concerning the potential for injury and teach safe hunting practices. No hunter in our study was known to be wearing a safety belt, yet it is recommended that *every* hunter wear a safety belt while ascending, descending, or sitting in a tree stand.[12] Hunters should also be educated to inspect trees carefully for weakened or dead branches, to let someone know where they will be hunting and when they will return, to avoid the use of alcohol or other drugs while hunting, and to get down from a tree stand when becoming drowsy. Hunters should also thoroughly inspect stands each season before use, checking for structural defects. This is an especially important precaution for homemade varieties. In one study, six out of seven falls due to structural failure of a tree stand involved self-constructed varieties of tree stands.[7]

It is noteworthy that only one of the injured in our study would have been covered by Oklahoma's mandatory hunter safety certification law (SB 448), since it does not require completion of a hunter safety education course for persons born before January 1, 1972.[12] While some older hunters voluntarily take the course, the data suggests that older hunters may especially need to be made aware of the unique risks for serious injury that tree stands may impose and be instructed in safe tree stand use and safe hunting practices in general. Tree stand safety instruction is included in the mandatory hunter safety class for younger hunters.

While no spinal cord injury in our study was related to unintentional discharge of a firearm while hunting, there have been other injuries reported elsewhere related to firearm discharge while ascending or descending from a tree stand, or while using a pull rope to transport a weapon to and from a tree stand.[6,9] Similarly, the Oklahoma Department of Wildlife Conservation reported that one recent injury in Oklahoma was related to discharge of a loaded firearm which was being lowered from a tree stand on a pull rope (personal communication, J. Peer). While pull ropes are the only recommended method of transporting weapons to and from tree stands,[12] hunters should be reminded to transport only *unloaded* weapons.

Finally, while regional differences in etiologies of injuries often go unobserved and unreported, this study is a good example of how ongoing statewide surveillance can work to identify the occurrence of repeating, localized patterns of injuries which might not be detectable in national data sets or one-time surveys. Regional surveillance may, in fact, be the *only* way small clusters of injuries are identified. Without such, the tragic reoccurrence of highly preventable injuries such as those in this report may never be stopped. Physicians and others dedicated to preserving the health of individuals can join in injury prevention efforts through reporting to the injury surveillance system and by disseminating injury prevention information to clients at risk.

Acknowledgment

The authors thank J.D. Peer and Steve DeMaso for providing hunting injury data and other information contributed to this report, and Nancy Kinman for preparation of the text and illustrations.

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The Authors

Cheryl Price, MPH, is an epidemiologist in the Injury Prevention Service, Oklahoma State Department of Health, Oklahoma City, Oklahoma

Sue Mallonee, RN, MPH, is service chief of the Injury Prevention Service, Oklahoma State Department of Health.

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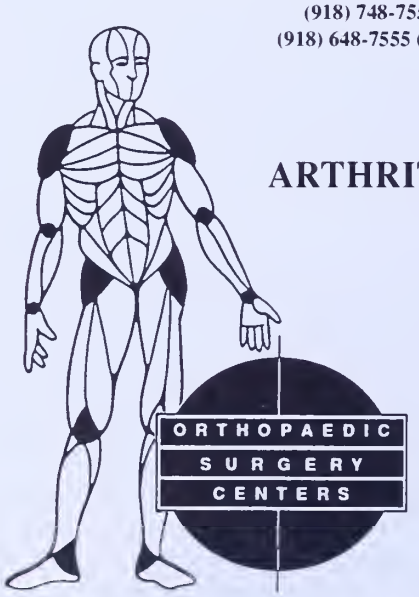


ARTHRITIS SURGERY CENTER OF OKLAHOMA

Norman L. Dunitz, M.D.	Mayo Clinic trained; Oklahoma pioneer in Joint Replacement development
Scott J. Dunitz, M.D.	Mayo Clinic trained; specializing in Joint Replacement Surgery and Surgery of Arthritis
James C. Slater, M.D.	Campbell Clinic trained; Full Fellowship Specialty in Arthritis Surgery; Harvard/Tufts

Shangri-La meeting is history as OSMA looks to the future

"OUR DAMNED DEMOCRACY," AMA President-Elect Robert McAfee calls it. "It's the AMA's greatest strength—and its greatest weakness."

Dr. McAfee was talking to delegates at an OSMA reference committee meeting in April. And for many at this year's Annual Meeting, he might well have been speaking of the OSMA.

The colorful three-day event at Shangri-La Resort bore a marked resemblance to a good old-fashioned political convention as delegates gathered to elect new officers, vote on resolutions and, in general, conduct the association's business. There were caucuses, campaigns, factions, and floor fights. And when it was over, as with all good compromises, no one was totally satisfied.

Still, representative democracy, the system organized medicine loves to hate, had its day. Every delegate had a voice; every delegate had a vote. The system, with all its shortcomings, worked. And the association emerged bruised but unbowed, its direction determined for yet another year.

AMA President-Elect Robert E. McAfee, keynote speaker at this year's meeting, calls for unity in the profession.

Dr. Alice McInnis (c), Oklahoma City, makes a pitch for her candidate to Dr. Mary Ann McCaffree, also of Oklahoma City, and Dr. Robert Phillips, Stillwater.

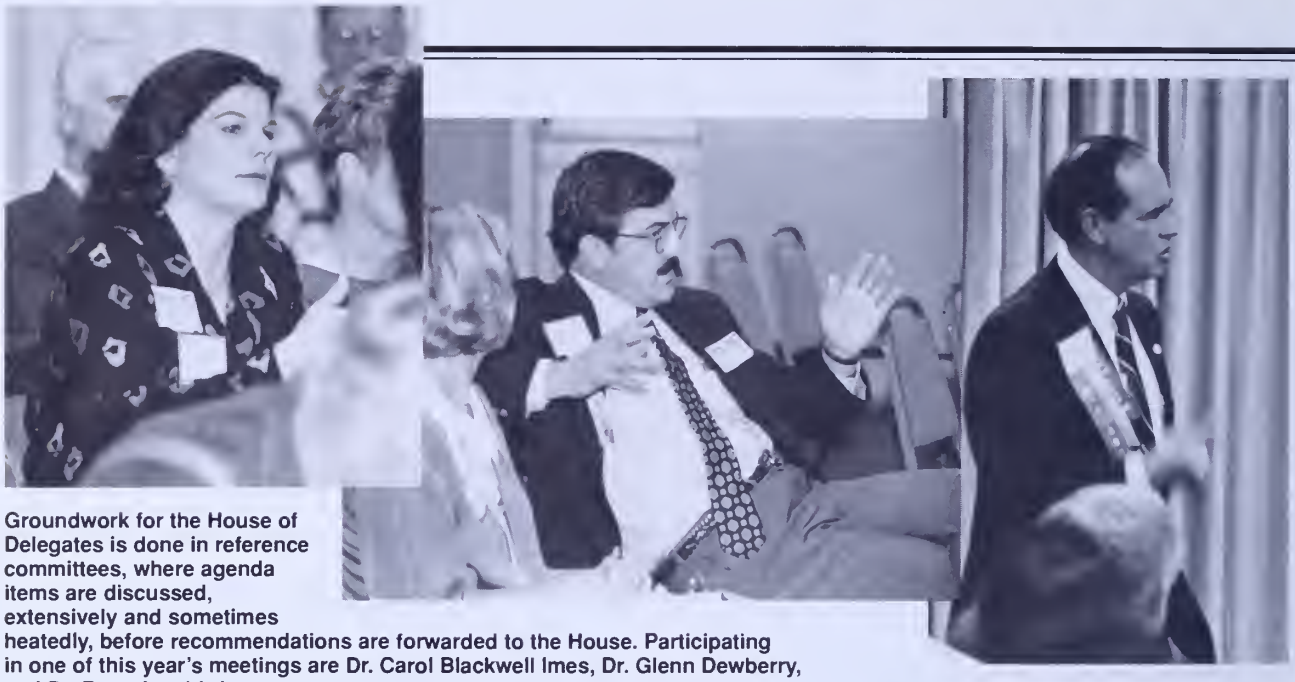
Election Results

Unlike many previous elections, this one featured several hotly contested races. Interest was high and the Saturday morning Candidates' Forum, usually sparsely attended, drew a standing-room-only crowd. Campaign buttons, stickers, flyers, candy, and even T-shirts appeared everywhere.

■ **OSMA.**— Leading the OSMA with President Jay A. Gregory, MD, a Muskogee surgeon, will be President-Elect Larry L. Long, MD, Oklahoma City surgeon; Vice-President David L. Harper, MD, Tulsa urologist; Secretary-Treasurer Carol Blackwell Imes, MD, Oklahoma City ophthalmologist; Speaker of the House Mary Anne McCaffree, MD, Oklahoma City pediatrician; Vice-Speaker Boyd O. Whitlock, MD, Tulsa internist; Chairman of the Board Douglas C. Hubner, MD, Tulsa pathologist; and Vice-Chairman Chester L. Bynum, MD, Norman radiologist.

■ **PLICO Board of Directors.**—Re-elected in contested races for the PLICO Board of Directors were William O. Coleman, MD, Oklahoma City; Joe S. Hester, MD, Muskogee; and David M. Selby, MD, Enid. Newly elected are Scott J. Dunitz, MD, Tulsa; and Michael J. Haugh, MD, Tulsa.

■ **AMA Delegates.**—Besting their opponents in races for AMA delegate slots were John R. Alexander, MD, Tulsa; Perry A. Lambird, MD, Oklahoma City; and Dr. Gregory. Winning seats as alternate delegates were Drs. McCaffree, Selby, and Whitlock. (cont.)



Groundwork for the House of Delegates is done in reference committees, where agenda items are discussed, extensively and sometimes heatedly, before recommendations are forwarded to the House. Participating in one of this year's meetings are Dr. Carol Blackwell Imes, Dr. Glenn Dewberry, and Dr. Perry Lambird.

Meeting: Election Results *(continued)*

■ **OSMA Board of Trustees.**—Elected in uncontested races for the OSMA Board of Trustees were Richard E. Martin, MD, Pryor, and alternate Douglas G. Cox, MD, Grove (Dist. I); Robert H. Phillips, MD, Stillwater, and alternate C. Mark Palmer, MD, Ponca City (Dist. II); Dr. Selby and alternate James S. Gerber, MD, Okarche (Dist. III); William T. Morris, MD, Woodward, and Ed L. Calhoon, MD, Beaver (Dist. IV); Frank K. Buster, MD, Cheyenne, and alternate James R. Rhymer, MD, Clinton (Dist. V); Fred M. Ruefer, MD, Muskogee, and alternate Bonnie J. Ashing, MD, Tahlequah (Dist. IX); Robert J. Weedn, MD, Duncan, and alternate Richard J. Boatsman, MD, Lawton (Dist. XIII); and Jeffry S. Lester, MD, Mangum, and alternate Joseph L. Krueger, MD, Altus (Dist. XIV).

Resolutions

Nowhere was the democratic process more in evidence than in the House of Delegates. Reference committee reports, with their recommendations for House action, drew delegate after delegate to the microphone with questions, comments, and motions.

Resolution 9, in particular, provided a demonstration of the system in action. Authored by Dr. Patrick Lester of Tulsa, the controversial resolution, which lifted the mandate that OSMA members be AMA members, was first aired thoroughly in reference committee. Supporters and detractors alike were given ample opportunity to address the issue before discussion was closed. The resolution then was sent to the House with the committee's recommendation that it not be adopted and that the Board of Trustees survey the entire membership for its views on the issue.

In the House, another lengthy discussion ensued. Motions were made, amended, voted down, and carried. Dr. Lester rose repeatedly, referring often to a copy of *Roberts Rules of Order*, to defend his views. Finally, after a voice vote proved inconclusive, he invoked parliamentary procedure to request a ballot vote on the issue. Ballots were distributed and the resolution, by then being considered apart from the rest of the recommendation, was defeated, 154 to 49.

A subsequent motion that the recommended survey be conducted within 60 days and the results provided to the membership within 120 days did not carry.

Ultimately the House approved the following amended version of the reference committee's recommendation:

> ...recommends that Resolution No. 9 not be adopted. ~~and~~ Recommends that the Board of Trustees of the OSMA be requested to conduct a written survey of all Association members and non-member Oklahoma practicing licensed medical doctors regarding their attitude about mandatory ~~AMA~~ membership and that the survey results be brought back to this House for informational purposes ~~next year.~~ at the next annual meeting of the House of Delegates.

This year's resolutions, their authors, and their disposition were as follows:

ADOPTED

■ **Substitute Resolution 1: Certified Audit.**— (Alice McInnis, MD, Oklahoma County Medical Society Delegation) [*See also Resolution 1 under Not Adopted*]

RESOLVED, That an audit of special procedures by a reputable firm be performed on the OSMA to confirm the sound practices of the OSMA and recommend proper financial controls.

■ **Resolution 6: Health Care Reform.**— (Glenn O. Dewberry, Jr., MD, Oklahoma City)

RESOLVED, That the Oklahoma State Medical Association endorses the concepts such as Medical Savings Accounts, Medical IRAs, requiring individuals to have a minimum level of health insurance coverage, tax credits for health care expenses, as well as the other common sense aspects of Senator Nickles' proposal, the "Consumer Choice Health Security Act"; and be it further

RESOLVED, That the Oklahoma State Medical Association encourages all Oklahoma physicians to become personally involved in educating fellow Oklahomans regarding the issues before us in this national debate, so that the people of Oklahoma will better understand the need to resist instituting government managed health care which some proposals advocate, and instead, support proposals which advocate expanding individuals' choices about health care and reducing government control.

■ **Resolution 11: Uniform Application Form.**— (Tulsa County Medical Society)

RESOLVED, That the Oklahoma State Medical Association support the development and utilization by all insurance plans and managed health care organizations of a uniform application, and be it further

RESOLVED, That the Oklahoma State Medical Association work with the Centralized Credentialing Collection Service of Tulsa County Medical Society to implement the acceptance of a uniform application; and be it further

RESOLVED, That this resolution be presented for consideration by the House of Delegates of the American Medical Association.

■ **Resolution 12: Centralized Credentialing Collection Service.**— (Tulsa County Medical Society)

RESOLVED, That the Oklahoma State Medical Association support and endorse the concept of centralized credentialing by publicizing it to health care organizations throughout the State of Oklahoma; and be it further

RESOLVED, That if the Oklahoma State Medical Association establishes a managed care organization, the OSMA's MCO contract with the TCMS Centralized Credentialing Collection Service to verify the credentials of the MCO's participating physicians.

■ **Resolution 13: Use of MD Designation After Retirement.**— (Jay A. Gregory, MD, Muskogee)

RESOLVED, That the Oklahoma State Medical Association petition the Board of Medical Licensure and Supervision for an immediate change in its rules to allow physicians to continue to use the designation "MD" after their name without the addition of any modifier words or symbols indicating practice status.

■ **Late Resolution 14: Vaccines for Children (VFC).**— (Sara Reed DePersio, MD, Oklahoma County Medical Society, Community and Mental Health Committee)

RESOLVED, That the Oklahoma State Medical As-

Above, OSMA Speaker of the House Mary Anne McCaffree, MD, explains a point of order to the delegates.

On the floor of the House of Delegates, Dr. Philip Mosca (r) and Dr. Clarence Robison express their opinions at the microphone.

sociation endorses the Vaccine for Children Program and encourages backing and support from the medical community throughout the State of Oklahoma; and be it further

RESOLVED, That the Oklahoma State Medical Association supports the efforts of the Oklahoma State Department of Health and the State Department of Human Services in making Oklahoma a Universal Vaccine State.

■ **Late Resolution 19: Legislative Investigation of OSEEGIP** [*sic*].— (Larry Hrdlicka, MD, General Surgeon, Claremore)

RESOLVED, That the OSMA ask the Oklahoma State Legislature to conduct an investigation into the contractual obligations of the OSEEGIP; and be it further

RESOLVED, That the OSMA ask the Oklahoma State Legislature to investigate the OSEEGIP's apparent capricious and ruinous conversion factor in implementing the RBRVS.

■ **Late Resolution 20: Economic and Professional Risk to Physicians in Hospital Merger and Closure Situations.**— (Clarence Robison, MD, Oklahoma City)

RESOLVED, That the membership be advised of these probabilities so that they may make appropriated financial arrangements to protect themselves through physician based managed care arrangements; and be it further

RESOLVED, That physicians be advised of the need to secure custody of their "personal 201 file" from a defunct institution in order to protect themselves from any adverse material that may have been placed there, either appropriately or inappropriately, with or without their knowledge, and with or without corroboration, so that such information does not fall into the hands of the general public; and be it further

RESOLVED, That the members be advised that severe limitations or denial of their previously enjoyed hospital privileges at the surviving institutions after merger or acquisition, may occur unless the medical staff is intimately involved in negotiating the surviving staff bylaws, and individual privileges of each member of the combined staff.

■ **Late Resolution 21: Economic Abuse of Physician by PHOs, HMOs, etc., Through Economic Credentialing (Bait and Switch Salesmanship).**— (Clarence Robison, MD, Oklahoma City)

RESOLVED, That the OSMA inform the public of the realities of Economic Credentialing; and be it further

RESOLVED, That the legal staff of OSMA provide advice and information to members who desire to join class action lawsuits against such entities who in this fashion abuse them and rob them of the value of their good name in the interest of "Health System Reform."

Above, OSMA Legal Counsel Ed Kelsay assists Comptroller Shirley Burnett with her Presidential Citation. Kelsay, associate executive director of the association, and Executive Director David Bickham were made honorary OSMA members in recognition of their combined 53 years of service.

Right, Wilson D. Steen, PhD, winner of the Donald J. Blair Friend of Medicine Award, expresses his appreciation to the association.

ADOPTED AS AMENDED

■ **Resolution 3: Signatures on Checks.**— (Alice McInnis, MD, Oklahoma County Medical Society Delegation)

RESOLVED, That two (2) signatures be required on all OSMA checks exceeding ~~$100.00;~~ $1,000.00; and be it further

RESOLVED, that the signature cards include ~~only~~ elected officers and CEO of the OSMA.

■ **Resolution 5: Continuing Medical Education.**— (Thomas N. Lynn, MD, Member, OSMA Accreditation Committee)

RESOLVED, That the OSMA ~~House of Delegates~~ Board of Trustees appoint a task force which would be charged to study and make recommendations regarding:

1. The reinstitution of a continuing medical education requirement to maintain membership in the Oklahoma State Medical Association and/or

2. Recommending to the State Board of Medical Licensure and Supervision that a requirement for continuing medical education be incorporated into the

requisites for relicensure by the Board; and be it further

RESOLVED, That such study be completed by and recommendations made at the spring House of Delegates meeting in 1995.

■ **Resolution 7: Prescribing Drugs by Non-Physicians.**— (Marcel Binstock, MD, Tulsa)

RESOLVED, That OSMA take a strong public stand against enactment of ~~this statute;~~ any proposed statute permitting the prescribing of drugs by non-physicians. ~~and be it further~~

~~RESOLVED, That the Governor, as the ultimate guardian of the interests of the people of this state, be urged to exercise his veto prerogative against enactment of this statute for the purpose of preserving public health.~~

■ **Resolution 8: Physician, Incorporated.**— (Robert Melichar, MD, Tulsa)

~~RESOLVED, That the Oklahoma State Medical Association conclude its separate efforts to create its own insurance company (IPA, MCO, etc.); and be it further~~

~~RESOLVED, that, in order to proceed as quickly and economically as possible to develop a truly statewide organization to control patient care, the Oklahoma State Medical Association provide complete support and endorsement to Physicians, Incorporated.~~

RESOLVED, That the House of Delegates encourage the boards of the MCO and IPA being created to initiate discussions with the Board of Physicians, Inc., for the purpose of developing the most effective mechanism for delivering high-quality patient care in a cost-effective manner, realizing the objective of creating a statewide, physician-owned organization.

■ **Late Resolution 15: Human Immunodeficiency Virus (HIV) and Sexually Transmitted Diseases (STD) Prevention in Adolescents.**— (Sara Reed DePersio, MD, Oklahoma County Medical Society, Community and Mental Health Committee)

RESOLVED, That the Oklahoma State Medical Association advocates prevention efforts to stop the spread of sexually transmitted diseases in adolescents. Prevention efforts should include effective Human Immunodeficiency Virus (HIV) and Sexually Transmitted Diseases (STD) and sex education as part of a comprehensive health curriculum for all students in grades 5-12 attending public schools in Oklahoma; and be it further

RESOLVED, that the Oklahoma State Medical Association recommends that the Youth Risk Behavior Survey (YRS) developed by the Centers for Disease Control and Prevention (CDC) be implemented by the Oklahoma State Department of Health in collaboration with the State Department of Education and the State Department of Mental Health and substance Abuse to gather baseline and follow-up data on health risk behaviors from students in grades 9-12. The data will be used to develop programs to address specific risk behaviors and to evaluate prevention efforts~~.~~; and be it further

RESOLVED, That OSMA recommend the Oklahoma State Health Department review the HIV, STD and sex education curriculum for appropriateness, content and accuracy.

Dr. Larry L. Long, in a successful bid for the office of president-elect, speaks at the heavily attended Candidates' Forum.

NOT ADOPTED

■ **Resolution 1: Certified Audit.**— (Alice McInnis, MD, Oklahoma County Medical Society Delegation) [*See also Substitute Resolution 1 under Adopted*].

RESOLVED, That a Certified Audit by a reputable outside firm be performed on the OSMA and all of its subsidiaries to confirm the sound financial practices of the OSMA and guarantee proper financial controls of the proposed IPA/MCO; and be it further

RESOLVED, that the Certified Audit will be presented to the House of Delegates with appropriate recommendations at an interim or future OSMA House of Delegates meeting in 1995.

■ **Resolution 2: Requirement of OSMA to Stay Within Budget.**— (Alice McInnis, MD, Oklahoma City, Oklahoma County Medical Society Delegation)

RESOLVED, That the Oklahoma State Medical Association cannot spend more than the approved Budget without approval by the House of Delegates.

■ **Resolution 4: Physician Recovery Program Loans.**— (Alice McInnis, MD, Oklahoma City, Oklahoma County Medical Society Delegation)

RESOLVED, That no more than $5,000 be loaned to any physician in the Physician Recovery Program; and no more than $2,500 to any OSMA staff member in a recovery program; and be it further

RESOLVED, That the loan must be repaid in full with appropriate interest, at a currently acceptable interest rate, within two (2) years of the loan.

Meeting: Resolutions Not Adopted *(continued)*

■ **Resolution 9: Deunification.**— (Patrick Lester, MD, Tulsa)

RESOLVED, That the Oklahoma State Medical Association cease its requirement that physician members also must be mandatory members of the American Medical Association; and be it further

RESOLVED, That effective January 1, 1995, and until the bylaws are changed to reflect the intent of this resolution, no physician shall be suspended or terminated from membership in the Oklahoma State Medical Association for failure to pay dues allocated for American Medical Association membership.

■ **Resolution 10: Victims' Insurance Fund.**— (Joseph L. McDonald, MD, Tulsa)

RESOLVED, That the Oklahoma State Medical Association endorse legislation by the Oklahoma State Legislature which would require the purchasers of new and used firearms to pay a percentage of their value into a victims' insurance fund; and be it further

RESOLVED, That this fund be allocated to pay for medical expenses of gunshot victims.

■ **Late Resolution 17: Domestic Abuse.**— (OSMA Perinatal Task Force)

RESOLVED, That the OSMA work to enact legislation to provide for the long term care and protection for victims of domestic abuse be they children, women, or men.

Referred to the Board of Trustees for Study and Report Back to the House of Delegates

■ **Late Resolution 16: Osteopathic Membership in the Oklahoma State Medical Association.**— (Donald E. Arego, MD, President, Comanche, Cotton, Tillman Medical Society)

RESOLVED, That the Oklahoma State Medical Association proceed with a revision of the Bylaws to allow the OSMA and county medical societies to allow Osteopathic physicians to become members of these organizations in accordance with all other existing membership requirements.

Rejected by the Board of Trustees, Not Considered by the House of Delegates

■ **Late Resolution 18: Composition of OSMA Board of Trustees.**— (William O. Coleman, MD, Chairman, OSMA Hospital Medical Staff Section)

RESOLVED, That the House of Delegates should see that this expertise [of medical staff members regarding the operation of a successful managed care organization] is utilized in the most productive manner by amending the Association's bylaws to provide that a representative of the OSMA Hospital Medical Staff Section should be designated a full voting member of the Board of Trustees.

MCO/IPA

In other action, the House approved the creation of a managed care organization and an independent practice association, as described in Report B of the Board of Trustees and recommended by Reference Committee IV. This came after Tom Garvey and Dan Boswell, of the consulting Garvey Group, presented their final report and Reference Committee IV met in an extended session to discuss the matter.

■ **MCO.**— The principles approved for the formation of the MCO as stated in Report B, are as follows:

✓ Intrastate stock corporation;

✓ Fifteen-member physician Board of Directors, all stockholders, elected by the shareholders;

✓ An Executive Committee of five members of the Board plus the Chief Operating Officer who will have a vote on the Executive Committee, but not on the Board;

✓ Board members will serve three-year terms (first board to be staggered by lot) and eligible to succeed themselves;

✓ Bylaws to require a majority vote of a quorum for the passage of any issue and a 3/5 vote of the Board to change any bylaws;

✓ Stock will be sold to raise capital. Only one class of stock will be offered;

✓ Only physicians (MDs and DOs) can purchase stock. (One share, one vote, per physician-stockholder.);

✓ Bylaws will contain a buy/sell agreement for stockholders who terminate their relationship with the MCO for any reason; and

✓ Only MCO shareholders can be IPA participants.

Note: The Interim Board (roster attached) shall include three (3) physicians selected by the Oklahoma Osteopathic Association (OOA) upon approval and ratification of the OSMA proposal by the OOA. The method for selecting the first permanent board is attached.

■ **IPA.**— The principles approved for the formation of the IPA are as follows:

✓ It shall be a not-for-profit corporation owned by its members;

✓ Membership available to physician members of the OSMA and OOA (MDs and DOs) upon payment of a membership fee;

✓ Membership may be terminated in accordance

with the rules at any time;

✓ An annual meeting of the membership shall be held in Oklahoma;

✓ One-third (1/3) of the membership constitutes a quorum;

✓ Bylaws amended or repealed by majority vote of members constituting a quorum;

✓ Bylaws require majority vote for passage of an issue and proxy voting is allowed;

✓ Board of Directors consists of eighteen (18) members—staggered terms of three (3) years with two (2) consecutive terms limit;

✓ Officers—President, Vice President, Secretary and Treasurer; and

✓ Only members may serve as officers or directors.

Note: The current interim board (roster attached) shall include three physicians selected by the Oklahoma Osteopathic Association upon approval and ratification of the plan by the OOA.

■ **Recommendations.**— The recommendations to the House of Delegates, which were approved by the House, are as follows:

✓ Confirms the interim boards attached hereto as the operating boards for the respective IPA and MCO. (Vacancies to be appointed by the president and appropriate inclusion of osteopathic physicians to be accomplished upon approval by the Osteopathic Association;

✓ Approves the formation of the MCO and IPA as proposed;

✓ Endorses the voluntary stock sale as the method of capitalization for the MCO with each unit of stock being sold for $4,000;

✓ Agrees to facilitate transitional funding as necessary and approved by the Board of Trustees;

✓ Approves the methodology for selecting the first Board of Directors (see attached plan).

[*On April 21, 1994, delegates at the Oklahoma Osteopathic Association's annual meeting unanimously approved a motion to "cooperate with the OSMA in developing a statewide Independent Practice Association (IPA) and Health Maintenance Organization (HMO) which would be available to respective members of the OOA and OSMA."*]

■ **Interim Boards.**— Members of the interim MCO board are Donald Crawley, MD, Stillwater; Eldon Gibson, MD, Shawnee; Joe Hester, MD, Muskogee; James Jones, MD, Duncan; William McCurdy, MD, Norman; Terry Dolan, MD, G. Lance Miller, MD, and Floyd Miller, MD, Tulsa; David Selby, MD, Enid; R.B. Winters, MD, Poteau; Ken Whittington, MD, Bethany; and Perry Lambird, MD, Larry Long, MD, Mary Anne McCaffree, MD, Phil Mosca, MD, and Mike Winzenread, MD, Oklahoma City.

Left, D.J. Flournoy, PhD, is this year's winner of the Leebron Award for best scientific paper published in the JOURNAL in 1993.

Right, Dr. Joe Crosthwait, stepping aside as leader of Oklahoma's delegation to the AMA, thanks the House for his resolution of commendation.

Members of the interim IPA board are Richard Boatsman, MD, Lawton; Billy Dale Dotter, MD, Okeene; Carl Critchfield, MD, and Jay Gregory, MD, Muskogee; Billy Irons, MD, Ponca City; Jon Axton, MD, Carol Blackwell Imes, MD, and Nick Knutson, MD, Oklahoma City; Ron Kreger, MD, Ponca City; Dennis McIntyre, MD, Enid; William T. Morris, MD, Woodward; Tim Smalley, MD, Stillwater; Bruce Storms, MD, Chickasha; Donna Brown, MD, Douglas Hubner, MD, and Boyd Whitlock, MD, Tulsa; and Jim Young, MD, Bartlesville.

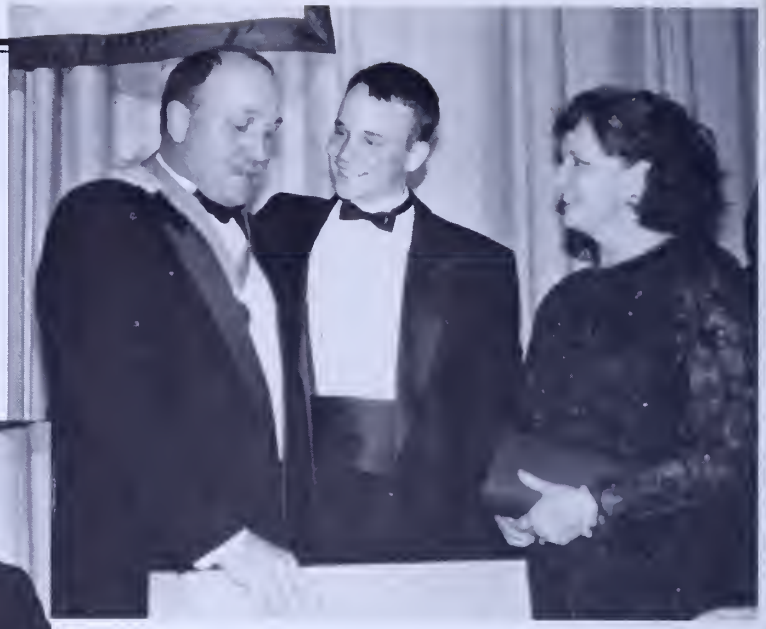
■ **MCO Board of Directors.**— The approved plan for electing the first permanent Board of Directors of the MCO corporation, as stated in Report B, is as follows:

✓ Prior to the 1st anniversary of the corporation, the Interim Board of Directors shall appoint a 5-member Nominating Committee each of whom shall be a shareholder, but not a member of the Interim Board;

✓ The Nominating committee shall prepare a list of a minimum of 15 shareholders as nominees for election as the Board of Directors of the corporation;

✓ The election for the first permanent Board of Directors shall be conducted by mail. Each shareholder will be provided an official ballot to fill out and return within 14 days of the original mailing. Write-in candidates will be allowed.

✓ The 15 nominees with the most votes cast at the annual meeting, will be declared duly elected;



At left, top, Dr. Jay Gregory (r), new OSMA president, is presented his old OSU football jersey by then-and-now team physician and friend, Dr. Donald L. Cooper.

Left, Dr. Gregory presents a plaque to his predecessor Lance Miller, thanking him for being "the right man in the right place at the right time."

Above, the newly inaugurated president receives congratulations from son, Eli, and wife, Edith. He holds a Presendential Citation presented to him by Dr. Miller.

Meeting: MCO/IPA *(continued)*

✓ The elected Board shall by lot, or otherwise as agreed by the majority, determine staggered terms of service of 5 years for 5 members; 3 years for 5 members and 1 years for 5 members.

Awards, Presentations, and Prizes

■ **Life Memberships.**— At its Thursday, April 14, meeting, the OSMA Board of Trustees approved Life Memberships for Joseph L. Martin, MD, A.J. Sands, MD, and James R. Geyer, MD, Oklahoma City; Orin J. Hake, MD, Stillwater; Ralph Simon, MD, Clinton; and John P. Skelly, MD, Tulsa.

■ **From the OSMA.**— Wilson D. Steen, PhD, was presented the Donald J. Blair Friend of Medicine Award during the Opening Session of the House of Delegates Friday morning. Unable to attend was Dr. Bertha M. Levy, retired Oklahoma City pediatrician, who was named Wyeth-Ayerst Physician Award for Community Service. A Resolution of Commendation was presented to M. Joe Crosthwait, MD, Oklahoma City, for his many years of service as delegate and chairman of the Oklahoma delegation to the ama.

■ **From AMA-ERF.**— Mrs. Darlene Young, secretary of the AMA Alliance, and Mrs. Mildred Taylor, president of the Southern Medical Association, attended the Opening Session in order to present checks from the AMA-ERF to Harold Brooks, MD, dean of the OU College of Medicine-Tulsa— $28,790.28 for the University of Oklahoma College of Medicine in Oklahoma City and $1,830 for the Tulsa branch.

■ **From the Journal.**— As announced previously, Dr. D.J. Flournoy was this year's recipient of the JOURNAL's Charlotte S. Leebron Memorial Trust Award for the best scientific paper published in the JOURNAL during the preceding year. Dr. Flournoy, a PhD, earned the special recognition by virtue of having authored one paper in 1993, co-authoring a second paper in 1993, and authoring or co-authoring a total of 23 papers in the JOURNAL since 1979.

The JOURNAL's Best Cover Photo Award went to Robert M. Smith, MD, Oklahoma City, for his October 1993 cover photograph of Red Rock Canyon. Dr. Smith was unable to attend the meeting and his award was presented to him in Oklahoma City.

■ **From the OU Alumni Association.**— The University of Oklahoma College of Medicine Alumni Association held its annual awards dinner Friday night. Honored were John M. Huser, Jr., MD, Private Practice Physician of the Year; Fred G. Silva, MD, Academic Medicine Physician of the Year; and Stanton L. Young, Amicus Medicinae recipient.

■ **At the Inaugural Banquet.**— At the Inaugural

PIAA releases results of latest malpractice claims study

Neurological and other birth-related injuries; cancer; fractures and spinal problems; and cardiac arrest and heart disease continue to account for a vast majority of malpractice claims against physicians, according to the Physician Insurers Association of America's (PIAA) most recent study of malpractice claims submitted to its ongoing Data Sharing Project.

The 5,340 claims related to these four areas account for 77% of total losses paid and 68% of all paid claims submitted since the start of the Data Sharing Project in 1985. The total indemnity paid in these cases totaled $1.2 billion, an average of $220,057 per claim.

These findings come from the Data Sharing Project's 17th reporting cycle, the six-month period that ended June 30, 1993. PIAA's Data Sharing Project is a system that collects specific demographic, payment, and loss causation information on malpractice claims reported by PIAA member-companies (including PLICO) participating in the Data Sharing Project: PIAA collects and analyzes this malpractice claims data semi-annually to help member-companies and their insured physicians reduce the incidence of patient injury and, thereby, reduce malpractice claims. PIAA strongly recommends against using this data for rate-making purposes.

Twenty-two of PIAA's 44 member-companies now participate in the Data Sharing Project. As of the end of the latest reporting cycle, more than 102,000 open and closed claims and suits were reported. Indemnity payments for these claims and suits totaled more than $3.8 billion on just over 29,000 paid claims.

The report provides statistics on the number of claims reported and dollars paid by medical specialty. Of the 24 specialty groups studied in the Data Sharing Project, obstetric and gynecological surgery have the highest number of claims reported (15,312, or 15%), with total indemnity payments of $899 million. General and family practice have the highest percentage of cases closed with a payment (40%). All specialty groups average 32.6%. Pediatrics is the specialty with the highest average payment per closed case, $206,200. This compares with the overall average of nearly $133,000. The average payment for pediatric claims has risen 38.6% since 1987.

The latest Data Sharing Report also shows that the

(continued)

Meeting: Awards *(continued)*

Dinnr Saturday night, OSMA Executive Director David Bickham and Legal Counsel Ed Kelsay received plaques granting them honorary memberships in the OSMA.The awards recognized 26 and 27 years of service, respectively. Also at the dinner, outgoing President G. Lance Miller presented Presidential Citations recognizing the service of OSMA Comptroller Shirley Burnett, OSMA President-Elect Jay A. Gregory, MD, and Dr. Crosthwait. Dr. Gregory then presented a plaque of appreciation to Dr. Miller.

■ **At the Closing Session.**— At Sunday's Closing Session, Dr. Larry L. Long, newly elected OSMA vice-president was presented with an oversized gavel to commemorate his decade of service as speaker of the OSMA House of Delegates. He was joined at the podium by the presidents under whose administrations he served: Drs. Amen, Crosthwait, Dotter, Dunitz, Eskridge, Funnell, Kamp, Lambird, J. McIntyre, R. McIntyre, and Miller.

■ **Golf Tournament.**— The Saturday golf tournament on the Shangri-La Gold Course was won by the foursome of Drs. Martin Woodall, David Thomas, B.R. Miller, and Joe Crosthwait, with a score of 59. Each of the four golfers received a gift certificate and a trophy. The tournament's hospitality cart was sponsored by Jeff Boyd and Verlan Raines of Administrative Service Corporation of Oklahoma City.

■ **From the Exhibitors.**— Delegates, spouses, and guests attending the Annual Meeting were encouraged to register at each exhibitor's booth for a prize drawing to be held during Sunday's Closing Session. W.H. Oehlert, MD, won a weekend at Shangri-La, and Barbara Jett, won a trip for two to London or Paris, provided by Seagrave Travel.

A Mont Blanc pen donated by Home Health Care Associates went to Dr. Mike McGee of Tulsa. A gift basket provided by Northeast Oklahoma Rehabilitation Hospital went to Dr. Bruce Storms and another given by American Transitional Hospital went to Dr. Doug Hubner. Dr. J.L. Kruger, Altus, won a pocket color television provided by Works & Lentz, attorneys.

Next Year

Next year's Annual Meeting of the OSMA House of Delegates will be April 7-9, 1995, at the Marriott Hotel in Oklahoma City. Any OSMA member may attend, but only duly elected delegates may cast votes.

PIAA Study *(continued)*

number of large loss claims has increased 10% since 1987. PIAA defines large loss claims as those with a payment of more than $100,000. Just over 10,000 claims (nearly 35% of the total paid claims) are in this category, representing total payments of $3.29 billion, or about 85% of the total indemnity payments. The percentage of large loss claims reported to the Data Sharing Project has increased to 35% from 27% in 1987.

The latest Data Sharing Project indicates that the following 10 specialties have the highest total indemnity:

Specialty	Indemnities (millions)	Average Payment	Approx. % of All Claims
Ob/Gyn	$899	$184,720	15%
Gen Surg	$471	$123,302	12%
Int Med	$428	$133,493	14%
GP/Fam	$404	$106,173	11%
Orth Surg	$343	$114,836	11%
Anesth	$227	$151,723	4%
Pediatrics	$169	$206,230	4%
Radiology	$146	$104,270	4%
Neurosurg	$129	$204,749	2%
Oph Surg	$92.5	$115,643	3%

Although plastic surgery wasn't in the top 10 specialties in terms of indemnity, it was in the top 10 specialties in terms of claims filed. During the reporting cycle, 3,761 claims, or nearly 4% of the total, were filed against plastic surgeons. The total indemnity attributed to this specialty was $85.4 million.

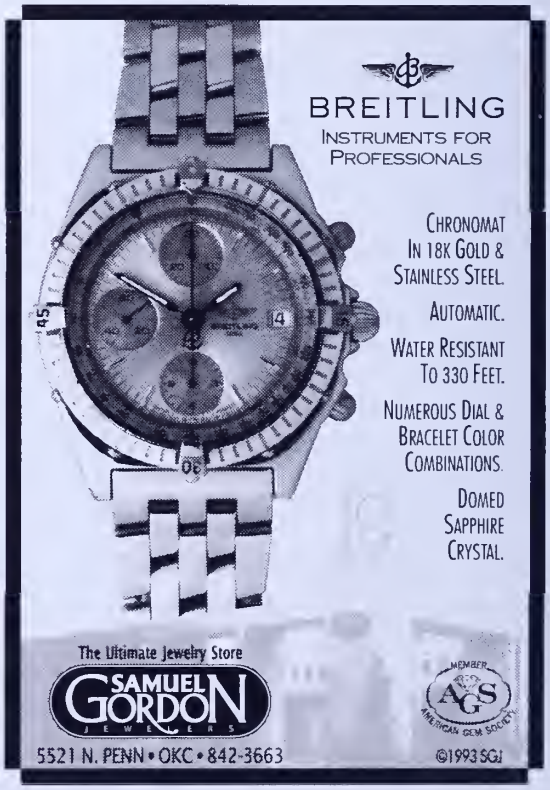
Other information gleaned from the latest report shows that the average indemnity per claim is now $132,927, a 2% increase from the $129,962 average indicated in previous reporting cycle, which ended December 31, 1992. More than one in five claims involve the death of the patient, and these cases account for 26% of the total indemnity paid. However, only 6% of all paid claims are rated with a severity code indicating a grave or fatal prognosis, but these cases account for almost 19% of the total indemnity paid and an average payment of over $395,000.

The majority of procedural accidents (66%) occur in a hospital, with over one-half of those occurring in the operating room. The physician's office is the second highest location of loss.

Just fewer than 15% of the claimants are age 18 or under, while nearly 18% are over age 60. Nearly 95% of the physicians involved in the claims are males and 78% of the insured physicians are board-certified. More than 74% of physicians involved in the claims attended a U.S. medical school, up 6% from five years ago.

A total of 60,005 of the 102,000 claims in the Data Sharing Project have been closed without an indemnity payment. The expenses associated with these claims total $566 million, an average of $11,300 per claim. Payments to defense attomeys account for 71% of the costs; payments to expert witnesses account for another 8.5%.

PIAA is an association of 44 physician-owned or controlled domestic malpractice insurance carriers that collectively insure about 210,000 (more than 60% of) independent practicing physicians in the United States. Among PIAA's primary objectives are to improve the quality of medical care for patients through loss prevention activities; improve the current medical liability system; suggest new malpractice claims resolution systems; and provide useful and accurate information on medical professional liability issues.



HEALTH DEPARTMENT

Vaccine management system tracks immunization records

One of the most important national health objectives of the U.S. Public Health Service for the year 2000 is to adequately immunize 90% of preschool children by their second birthday against vaccine-preventable diseases. In an effort to achieve this goal, the Oklahoma State Department of Health (OSDH) is implementing a computerized immunization tracking program (Vaccine Management System) at the local agency level. The Vaccine Management System was developed by the immunization division of osdh and is now available to private immunization providers at no charge. The cost for similar software developed by private firms ranges up to $2,500 per user license.

The Vaccine Management System is a menu-driven program that allows immunization tracking, client recall, and vaccine inventory management. The system's reporting capabilities include printing mailing labels for clients that are found to be "coming due" or "overdue" for immunizations, client immunization records, and inventory reports. Ad hoc reports may be created with the use of report-writing software capable of accessing dBase III–compatible files. This is a DOS-based program and is capable of functioning on a stand-alone PC as well as a local area network.

This software has been developed with the future in mind. Information collected through the Vaccine Management System will become part of a centralized immunization registry to be developed by OSDH. This statewide database will maintain children's immunization records, including whose who receive immunization from a variety of sources. Vaccine providers will be able to access the OSDH central registry for a child's immunization history so appropriate vaccine can be given without the medical staff attempting to reconstruct the child's previous immunization history.

Statewide immunization registries, such as the one planned by OSDH, are being heavily promoted by the Centers for Disease Control. All 50 states are expected to have immunization tracking systems in place by September 30, 1999, enabling states to easily exchange immunization information.

Immunization providers interested in obtaining more information about the Vaccine Management System may contact the Immunization Division of the Oklahoma State Department of Health at (405) 271-4073.

LETTERS

Discussion continues on Annual Meeting issues

To the Editor: After the state meeting at Shangri-La this year, I couldn't help but think of the deunification issue in a different light than ever before. The unified status of the OSMA and the AMA represents a hurdle over which many are not willing to jump to belong to OSMA. Indeed the AMA is an organization that is a hindrance to the strength of the OSMA, as many will not join the OSMA because of the affiliation with this ever-compromising, unprincipled national organization. It remains to be seen how many currently belonging to the OSMA will drop their membership after the delegation again defeated deunification.

Listening to AMA delegate candidates jockey for position by touting who they knew on the AMA board of directors was a real shock. This organization operates like the U.S. Congress—where who one knows matters more than ideas. To limit AMA delegate posts to former OSMA officers was wrong and an elitist move. Term limits are in order for the AMA delegates and these positions should be open to the membership of the OSMA without restrictions.

A survey regarding deunification is forthcoming and I would encourage all in support of deunification to send letters to OSMA officers and to the OSMA JOURNAL for publication. As divisive as the issue of deunification is, I am surprised and disappointed that the OSMA JOURNAL printed Dr. Lambird's comments regarding deunification without the opportunity for an editorial summarizing the issue from the pro-deunification standpoint, particularly since Dr. Lambird's comments were printed at the expense of the membership.

One delegate commented that the vast majority of the physicians in his district were for deunification but that as their representative he hoped that the resolution to deunify would be killed by amendment so that he would not have to discharge the unpleasant duty of voting as his constituency

(continued)

Worth Repeating

Hillary hears from Oklahoma City 'ghostbuster'

Dear Mrs. Clinton:

I was disappointed that I could not address you in Muskogee on April 8, 1994. I was invited in my capacity as President-Elect of the Oklahoma Society of Internal Medicine and we literally received about 16 hours notice. I had prepared some thoughts for you but unfortunately the time allowed for questions and answers was too short.

I make these comments wearing several hats: namely that of a physician, providing care to about 1,000 patients; a father of two; a potential recipient of care; a citizen of the United States; and a small business owner.

My patients seem to be very concerned about three issues: (1) the bureaucracy and the complexity brought about by the alliances, (2) the potentially explosive cost of health care reform, and (3) the fear of loss of quality of care. I would like to elaborate on these points.

First in your comments, you specifically said that you see the alliances "only as a pass through of the monies to buy insurance." If that is all the alliances will do, that is, behave as purchasing cooperatives, then I and my patients have no problems with them. It is the powers that are given to the alliances and all the bureaucracy that they entail when performing other functions as outlined in the President's plan that brings concern to our patients. Our patients want a simple plan that works for the majority of them. Alliances have numerous problems, not the least of which is that they are bound by artificial state lines. There are many instances of patients seeking care in another state, simply because it is more convenient to cross the state border than to travel across the mountains or the state to get to the local center of excellence.

Second, and now as a citizen who is extremely concerned about the national debt and about any government spending that would make the debt even worse, and as a father of two children who will almost certainly never be able to borrow money to achieve the American dream and buy a house, I beg you to consider the issues of cost. Even without having to look thirty years down the line, the current government debt makes America hostage to serious problems. By 1996, we could end up like Russia, Poland, Brazil, and Italy if we do not bring our spending under control. This is why incremental solutions in health system reform are best. You have discussed portability of insurance, abolition of the pre-existing condition exclusion, and gradual coverage of true preventive services, and we are 100 percent in favor of such small insurance reform steps. Anything beyond that could place a burden on small business and government that will either close small businesses by the droves or bankrupt the government further. It is worth remembering that small businesses provided 100 percent of new jobs between 1987 and 1992 and that they represented 99.7 percent of all employers in 1990. I therefore beg you to consider very seriously the potential negative economic impact of a massive government spending program. This is not to take away from your efforts and from your intent to provide insurance and health care coverage for the 37 million uninsured. I just think, together with many of my patients and colleagues, that we can get there but less radically and more slowly.

(continued)

Letters (continued)

directed him. Are the OSMA and the delegates sent to represent the physicians in their districts functioning in a representative manner? Should we operate as a republic or as a democracy, as the National Federation of Independent Business does? If issues before OSMA were settled by a majority vote of the members, would OSMA appear to be less of a PAC, lobby group representing special interests at the expense of patients and business in Okla., and more representative of what is good for all?

Dr. Patrick Lester deserves much credit for the hard work he has done (and I think will continue to do) to make this OSMA and AMA more responsive, representative and accountable to its membership.

—G. Keith Smith, MD
Oklahoma City

Note: Dr. Smith attended the meeting as a voting delegate for Oklahoma County. Dr. Patrick Lester, Tulsa, authored the deunification resolution.

Part of the JOURNAL's mission, as stated annually in its report to the OSMA Board of Trustees and House of Delegates, is "to serve as an open forum for the exploration and discussion of issues vital to the physicians of Oklahoma." Readers are encouraged to express their opinions in this column. Letters should be addressed: To the Editor, JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION, 601 Northwest Expressway, Oklahoma City, OK 73118.

—Ed.

Ghostbuster *(continued)*

Finally, and most importantly, I would like to address the issue of the fear of my patients and colleagues about the loss of quality of care. Excellence has a price. We have, however, confused available and abundant technology with high quality care; the best care is that delivered by caring, thorough, compassionate physicians in counselling on preventive health, in screening for common diseases that place a high burden on society, and in treating common and treatable, if not, curable disease. By prohibiting balance billing, the government will push excellent physicians into networks where quality will not be rewarded. Those physicians will not be able to maintain the habits that made them excellent to begin with, whether it is continuous medical education or introspection and good bedside manners. Banning balance billing will snuff out private fee-for-service solo practice, one of the true pillars of excellence of medical care: we are there when our patients need us, they know us, they know our phone numbers, they know our office address, they know what is in their medical record. We are there to allay their fears. We are their ghostbusters, if you wish. We walk them through the health system, a very frightening place to be, as you said in Muskogee and elsewhere. Whatever you do, please don't snuff out private solo practice. I have enclosed handwritten original petitions by my patients, who were in and out of my office in the last two weeks prior to my visit with you. I only see eight to ten patients a day and I certainly do not want to compromise thevery high quality of care that I provide and have provided for the last ten yearsbecause the government prohibits balanced billing.

The President recognizes that the practice expense component of the Resource Based Relative Value Scale Formula underestimates the true cost of rendering medical services; if you want to preserve private solo practice and the quality that stems from continuity of care, and if you want to save health care dollars in the long run, please make sure that all evaluation and management services, whether rendered by primary care doctors or by specialists, are reimbursed fairly and adequately.

Finally, Mrs. Clinton, I would like to thank you for your tireless efforts and for your high-minded intent to improve the health system in America. There is no doubt that it needs improving. You and your husband have spoken eloquently to the subject. We are certainly tired of lying to insurance companies and pretending that our patients don't have depression or diabetes or other so-called uninsurable illnesses. We welcome your efforts to make those conditions coverable and reimbursable. We just want to make sure that when it is over, our system continues to be one where excellence is available, and where the majority of patients are covered and get very adequate, if not very good care. Thank you for your attention. I look forward to your reply or to one of your aides' answers.

—S. A. Dean Drooby, M.D.
Oklahoma City

In Memoriam

1993

John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13
Earl Mathiews Woodson, MD	February 20
Tom Lamar Johnson, MD	March 5
Orville Main Rippy, MD	March 11
Minor Elliott Gordon, MD	March 14

Deaths

Tom Lamar Johnson, MD
1923 - 1994

Tom L. Johnson, MD, OSMA Life Member, died March 5, 1994, in Oklahoma City. Dr. Johnson was born in Houston, Miss., and grew up in Beaumont, Tex. During World War II he served in the Pacific Theater with the U.S. Navy. Following the war, he attended Southwestern Medical School in Dallas, earning his MD degree in 1950. He completed his residency in ophthalmology at Washington University School of Medicine in St. Louis. Dr. Johnson established a private practice in Oklahoma City after completing his education. Later he was appointed an assistant clinical professor at the University of Oklahoma Health Sciences Center.

OSMA toll-free number
1-800-522-9452

Classifieds

Classified ads are 50 cents a word, with a minimum of $25 per ad. A word is one or more characters bounded by spaces. Box numbers will be assigned upon request and will add 6 words to the total. Payment must accompany all submissions. Orders will NOT be accepted via telephone or fax. Mail ad with payment to OSMA Journal, 601 Northwest Expressway, Oklahoma City, OK 73118. Deadline is the first of the month preceding the month of publication.

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LITTLE ROCK, AR - FP partner needed for busy outpatient practice in Sheridan. 30 minutes from Little Rock. Beautiful facilities, full range of service. Above average first year of salary plus production bonus. For more information contact Christine Ross, R.N., 800-776-5776.

Oklahoma: Enjoy practicing Emergency Medicine in the great state of Oklahoma. Directorships and independent contractor practices are available now. No matter where you live or where you want to practice, we have a place that might be just right for you. Competitive remuneration, flexible scheduling,and a network of other dedicated physicians are just a few of the aspects of being associated with Coastal. For more information on Oklahoma opportunities, please contact Cheryl Grimm at 1-800-745-5402 or send/fax your CV to Coastal Emergency Services of Dallas, Inc., 3010 LBJ Freeway, Suite 1300, Dept. SJE, Dallas, TX 75234. FAX: 214-484-4395.

UROLOGIST, OB/GYN, FAMILY MEDICINE and ENT physicians needed for Southern Plains Medical Center, 33 physician multispecialty group in Chickasha, OK. Located 35 miles southwest of Oklahoma City on Interstate 44 in a family oriented college community of 20,000 with service area of 110,000 population. Outstanding medical center with ambulatory surgery center. 156 bed hospital next door. Excellent guarantee and benefits. Contact Jeanie Bledsoe, Recruiter, or Danny Vaughan, Administrator, 2222 Iowa, Chickasha OK, 405-222-9583 or 405-224-8111.

OSMA Physician Recovery Program

(405) 360-4535

THE OFFICE OF HEARINGS AND APPEALS (OHA) of the Social Security Administration is accepting applications from Board-Certified Physicians for consultant positions. Services will consist of expert testimony in social security hearings. For further information, please call or write Gwen Dickerson at OHA/SSA, 1200 Main Tower, Room 1135, Dallas, TX 75202 (214) 767-9401.

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1044 S.W. 44th St., Suite 210
Oklahoma City, Oklahoma
(405) 235-0040

NORMAN OFFICE
950 North Porter, Suite 101
Norman, Oklahoma
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Senior Consultants:
George S. Bozalis, MD
George L. Winn, MD†

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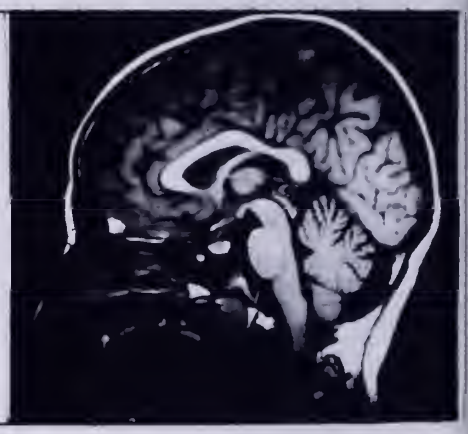

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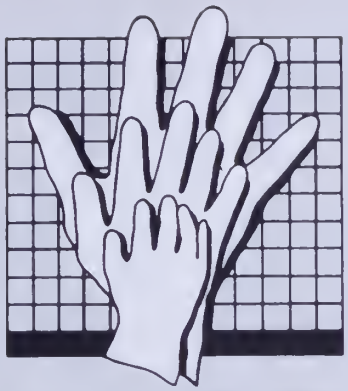

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THE LAST WORD

■ **JOURNAL Editor M. DeWayne Andrews, MD,** Oklahoma City, professor of medicine and chief of the General Internal Medicine Section at the University of Oklahoma Health Sciences Center (OUHSC), has been awarded one of the university's top distinctions, the David Ross Boyd Professorship. Named after the university's first president, the professorships were first awarded in 1944. They now carry a $6,000 award in the year of selection.

Dr. Andrews joined the OUHSC faculty in 1976 as assistant professor of medicine and in 1982 was named assistant dean for graduate medical education. He served as director of the residency program, Department of Medicine, from 1978 to 1984, and was named professor of medicine in 1988. He is also chief of staff at University Hospital.

A 1970 graduate of the OU College of Medicine, Dr. Andrews is governor-elect of the Oklahoma chapter of the American College of Physicians, a member of the American College of Physician Executives, and is on the OSMA Board of Trustees. He has been a JOURNAL editor since 1992.

His previous awards include the Outstanding Medicine Faculty Award, Department of Medicine Residents, from the OU College of Medicine in 1992, and the OU Aesculapian Award for excellence in teaching clinical sciences, 1989.

■ **The American Medical Association's Department** of Health System Reform Policy Analysis is looking for examples of physician-nurse professional collaboration. Examples will be used to examine and develop practice and payment profiles regarding nurses and physicians. Send examples of your professional experiences to Marcia Moore, JD, AMA, Department of Health System Reform Policy Analysis, 515 North State, Chicago, IL 60610, or fax material to (312) 464-5836. For details on the study, call 1-800-262-3211, ext. 4710.

■ **Tulsans Harvey J. Blumenthal, MD, and** Roger E. Wehrs, MD, have been named to the second edition of *The Best Doctors in America,* published by Woodward/White, Inc.

■ **The AMA is offering a free comparative** analysis of five health system reform proposals recently introduced in Congress. The report analyzes the Nickles/Stearns, Rowland/Bilirakis, Stark, Gramm, and Dingell reform proposals. Charts compare plans with AMA policies by coverage, mandates, choice, insurance, liability, antitrust, taxes, financing, workforce, and Medicare/Medicaid provisions. Copies are available by calling (312) 464-5463 or (312) 464-4774.

■ **Tulsa physicians are reminded of the Retired** Physicians Lunch and Speaker Meeting held the first Monday of each month. The group gathers at 11:30 AM in Dining Room 4, St. John Medical Center. For more information contact Dr. Mel Swafford, (918) 747-1155.

■ **"Violence as a Health Issue" is the title of a** seminar to be held October 29 in Tulsa. Sponsored by the Tulsa County Medical Society, Mayor's Task Force on Family Violence, and University of Oklahoma College of Medicine-Tulsa, the meeting will examine the abuse of children, the elderly, and other forms of domestic violence. Physicians are required by state law to report suspicion of battery: "Any physician examining, attending, or treating the victim for what is suspected to be criminally injurious conduct shall report the matter at once to the nearest appropriate law enforcement agency in the county wherein the criminally injurious conduct occurred."

■ **The Southern Medical Association's 88th** Annual Scientific Assembly will be held November 2-6, 1994, in Orlando, Fla. AMA Category I credits will be awarded. The meeting will feature a multispecialty format with specialty section meetings, specialty- and practice-related postgraduate courses, and scientific and technical exhibits. The registration hotline is 1-800-423-4992 or fax (205) 942-0642.

■ **Approximately three-fourths of Americans** would rather pay more and be able to select their personal physician, get care right away, and use a particular hospital, than pay less and have less flexibility. So say survey data released recently by the AMA. The proportion willing to pay more to choose their own physician increased to 81% this year, up from 76% in 1993. Fifty-seven percent of Americans would rather pay more out of pocket to private physicians and hospitals than pay higher taxes and have the government in charge of health care delivery. Complete survey results appear in the May 2 *American Medical News.*

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Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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Fourth of July fireworks brighten the night sky above Chickasha's Shanoan Springs Park.

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EDITORIAL

Ray V. McIntyre, MD

Who Has Guns?

In an America that has lost its equanimity in controlling crime, Congress has passed the Brady Bill, and is debating the definition of a firearm. The judges are now considering warrantless searches for guns. It seems that the deteriorating ability of our criminal justice system to protect the U.S. citizen has flustered the legislature and judiciary into a posture that may change the citizen's ability to self-defend. The Constitutional foundation stone of citizen self-defense was ratified December 15, 1791:

Article II

A well regulated militia, being necessary to the security of a free State, the right of the people to keep and bear arms shall not be infringed.

The Constitution of the United States was drafted with an economy of words that constricted the document to the basic principles of the relationship of the citizens to the government. Thus it was with the Second Amendment about weapons, reproduced here in its entirety. Presently the validity of the amendment is the focus of debate as our nation reacts to a spate of firearm deaths.

There is a widespread movement in the United States to restrict the possession of weapons to the police and the Armed Services as a means of reducing firearm deaths. It is said that America has a passion for violence and that Article II is no longer appropriate, as conditions have changed from the colonial times of origin. No one needs a firearm in today's world, they say, and the Second Amendment should be ignored and the guns controlled.

It is true that the world is different in many ways from colonial times. Instead of marauding frontier renegades we now have unchecked urban riots; instead of a British Army of occupation we now have universal susceptibility to muggers, burglars, and carjackers. The capability of the police and the criminal justice system to protect the ordinary citizen from raid and murder is probably less today than when Article II was written, and government oppression of citizen liberty is no less onerous now than it was in 1791. America has no more "passion for violence" than any other tribe of humans; we do have 24-hour news broadcasts into every home that repeatedly detail every violent act occurring anywhere. Now, as in 1791, every citizen should have the right of self-defense.

The term *militia,* used in the Second Amendment, means "every able bodied man between the ages of 18 and 45 who is not in the army." Thus we can see that it was the clear intent of the Founding Fathers to establish a prohibition on the government's ability to disarm the citizens. It is also clear that although the citizen could own and carry firearms, the manner of use was under government regulation .

Let us rewrite Article II into today's idiom:

A safe and secure citizenry being necessary to the welfare of a free State, the right of the people to own and carry firearms shall not be infringed.

Then let us, as a society, consistently punish the criminal use of weapons and also set out to teach our young people, both men and women, the safe and responsible use of firearms. Just as we use driver's education courses to teach the safe use of automobiles, so it is that everyone should become knowledgeable and responsible in the ability to use that self-defense tool known as a firearm.

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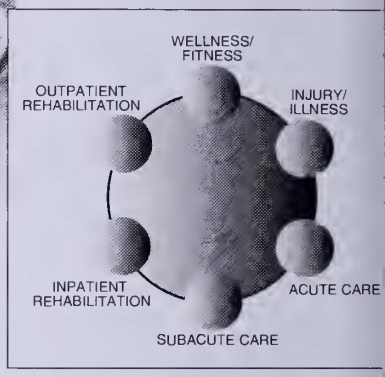

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President's Page

Jay A. Gregory, MD

Do We Have It Together, or What?

uni·ty 1. The quality or state of not being multiple; oneness. **2.** A condition of harmony; accord. **3.** Continuity without deviation or change (as in purpose or action). **4.** The quality or state of being made one; unification. **5.** A combination or ordering of parts in a literary or artistic production that constitutes a whole or promotes an undivided total effect; also: the resulting singleness of effect or symmetry and consistency of style and character. **6.** A totality of related parts; an entity that is a complex or systematic whole.

Why would I be writing to you about unity? Perhaps it is that we do not understand the meaning of the term. Perhaps we are on the verge of demonstrating the antithesis of unity, or **dis·uni·ty** A lack of unity; esp: dissension.

What are some of the elements that lead to disunity? Do we exhibit some of those characteristics? We have so many conflicts: Between the "good ol' boys" and the "young and the restless"; between primary care groups and specialty groups; between male and female; between those who are in power and have authority, and those who dream of possessing power and authority; between the status quo and the cries for change. And personal conflicts, between being a healer of the sick, available at a moment's notice when our patients call, and trying to be a parent and providing for our families.

Pogo was correct when he said,"We have met the enemy, and he is us." One of our national leaders recently said, "We have circled the wagons of medicine, and now we are shooting at each other." Dr. George Ladd, addressing the Muskogee Regional hospital staff, said several years ago, "Whether you like me or not, I am your best friend."

During the past few weeks I have sensed the pain, the agony, the helplessness of physicians faced with the reality of losing their practices and selling out to an entity which guarantees a salary and a set of benefits they were unable to provide for themselves. I feel the despair of those who argue for change but have no "power base" from which to leverage change. I feel the hurt of those who have preserved the status quo because they believe it is best for those of us who will follow.

I was under the impression that it would be only moderately difficult to lead your medical society this year. I thought I was experienced and had spent enough time learning the system to prepare me for this task. But when I was exposed to and faced with the realities that exist within our own house of medicine, I might easily have said these problems are insurmountable; these problems contain so much prejudice, anger, and false perception that no one can change them.

I do believe that while no *one* person can make an effective change, *groups* of you, taking very serious, in-depth looks at the problems and discussing or debating the issues in a civil and respectable manner can, via the democratic process, arrive at good solutions which will serve your patients and our association well.

Remember that "good docs, given good data, will make good decisions." Look yourself in the mirror and ask yourself, "Am I a good doc?" Always challenge the data. Go to the source; seek out the truth. You owe it to yourself not to be influenced by rumor and innuendo. Use the democratic process so that you and your colleagues arrive at good decisions. Then, regardless of the outcome of that process, support those decisions which were made on behalf of the whole.

None of the above can be accomplished alone. I need your help. Through a process of unifying all our efforts toward a common goal, we can and will become a stronger association. We can and will, through unity of purpose, make Oklahoma a better place in which to live by improving the health of all Oklahomans.

No one part is greater nor more important than the whole. It will take all of us, working together, to overcome the problems within before we can ever effectively beat back the outside challenges.

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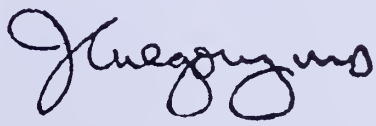

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SCIENTIFIC

Incidence Patterns of Immunogenetic Diseases in the North American Indians

Thomas E. Acers, MD; Ann Acers-Warn, MD

Epidemiologic studies have defined a number of disease susceptibility patterns based on host-environment interaction. The antithetical approach of studying disease resistance patterns has been utilized less frequently.

This preliminary data report describes the incidence of certain immunogenetic diseases in North American Indians for "internal comparison" of tribal population groups residing in disparate geographic areas and for "external comparison" with incidence patterns of Caucasian populations residing in the same geographic areas.

The preliminary nature of the data precludes any conclusionary statements.

Anthropologic and epidemiologic studies of certain diseases have defined a number of disease susceptibility patterns based on host-environment interaction. The antithetical approach of studying disease resistance patterns has been utilized less frequently. A unique model combining these two analytical approaches exists in certain geographic areas of the world where native and "immigrant" populations are cohabitant and yet often demonstrate striking disparity in their disease susceptibility and resistance patterns.

Whether these disease patterns are now changing due to an evolving heterogenicity secondary to "gene flow" or to sociocultural changes or both, is unclear. The Law of Biological Evolution states that "as culture evolves, the biological variability between human populations declines." Recent gene frequency analysis tends to validate this theory until a negative correlation with latitude is introduced. With control of this variable, other correlations between heterozygosity and sociocultural complexity became essentially nonsignificant.[1-3] Perhaps these same interrelated factors of heterozygosity, socioculture complexity and latitudinal habitat are now being manifested by changing disease patterns within our own Amerindian population groups.

As new disease entities occur, and with more sophisticated diagnostic technology and improved data collection methodology, changing patterns of disease occurrence may be expected. Historical (anecdotal) information may not be statistically valid but may be the only comparison base available in our more underserved nations and population groups.

The purpose of this study is first, to present data on the incidence of immunogenetic diseases in the North American Indians for an "internal comparison" of tribal population groups residing in disparate geographical areas. Then, to provide an "external comparison" of incidence patterns with the Caucasian populations residing in the same geographic areas.

This report is a review of the preliminary data acquisition and is recognized to be incomplete. It is based on the index of IHS hospital diagnostic codes which potentially creates an "across the board" underestimation of specific disease incidence rates. It is not intended to be conclusionary in any way, but more in the form of a preliminary data collection report (Table 1).

Direct correspondence to Thomas E. Acers, MD, 608 Stanton L. Young Boulevard, Oklahoma City, OK 73104.

Table 1. I.H.S. Area Populations

I.H.S. Area	Major Tribal Nations	Population (1990)
Aberdeen	Sioux, Cheyenne Blackfeet, Crow	75,000
Alaska	Athabascan tribes: Tlingit, Haida and Koyukon. Alaskan Eskimo (Inuit) and Aluets	86,000
Albuquerque	Apache, Pueblo, Navajo	66,000
Bemidji	Sauk & Fox, Winnebago, Kickapoo	61,000
Billings	Sioux, Cheyenne, Crow, Arapaho	47,000
Navajo	Navajo	181,000 (N. Mex-Ariz)
Oklahoma	Cherokee, Creek, Choctaw, Chickasaw, Seminole, Kiowa, Shawnee, Comanche, Osage, etc.	257,000
Portland	Shoshone, Paiute Nez Perce', Spokane Yakima	120,000
Phoenix	Apache, Pueblo, Hopi, Zuni	101,000

Multiple Sclerosis

Multiple sclerosis (MS) is an acquired, environmentally related and possibly transmissible (infectious) disease of unknown etiology which may lead to progressive neurological dysfunction, blindness, and death.

It appears to be a multifactorial disease process involving the interaction of genetic, environmental, infectious and immunologic factors .

Since World War II there have been more than 200 studies designed to study the natural history and geographic distribution of MS. These (prevalence surveys) have defined the high frequency zones of MS extending from 43° to 65° north latitude in western Europe, from 37° to 52° in the northern United States and Canada and from 35° to 50° southern latitudes in Australia and New Zealand.[4-11]

During the past two decades the most prevalent etiologic theory involves the interaction of viral infections and the host immune system. Antibody assay analysis of MS serum and cerebrospinal fluid has provided indirect evidence of viral infection with significant differences between the MS and control groups, including: higher serum antibody titres to measles virus in the MS group and a higher proportion of MS patients with CSF antibodies to measles, rubella, and vaccinia viruses.[12-19]

The more sensitive radioimmunoassay methods demonstrate the CSF IgG and the IgG/albumin ratio are both increased in the MS group. If methods for oligoclonal band detection and CSF IgG/albumin ratios are used, 95% of MS patients will demonstrate abnormalities but the biological specificity of this is still unknown. With the rapid DNA amplification provided by the polymerase chain reaction process, it may soon be possible to detect more direct evidence of a specific virus infection.

Genetic susceptibility has also been implicated as a disease risk factor, especially in the inhabitants of the northern United Kingdom. HLA-A3, B7 and D2 have been identified as specific risk combinations in the MS groups versus control studies, while kindred-migration studies in South Africa, Israel, and Australia have produced inconsistent data. Current twin studies involving "synergistic gene analysis" to identify specific genetic markers of predisposition may add to this area of information in the near future.[20-24]

An epidemiologic paradox exists though, in that certain aboriginal population subgroups residing in the high prevalence MS zones demonstrate almost complete resistance to developing MS. These include the North American Indians, Eskimos, and Aluets as well as the New Zealand Maoris and Australian Aborigines. Whether this is due to genetic or exposure factors is unclear.

Another distinctive ethnic population group living in a high risk MS zone and displaying a similar resistance to developing MS is the Hungarian Gypsy. Recent studies suggest a specific genetic protective factor in the absence of the Dqw6 fragment of the DR2 antigen, the presence of which is most frequently found in their Caucasian counterparts with MS.

Relative social isolation may allow these population groups to conserve certain immunogenetic singularities of a protective nature.

The factor of "gene flow" between native populations and nonnative (European) populations may be important in that one would expect that individuals with both native and European ancestors would be more susceptible to MS than those with only native ancestors. Blood quantum comparison may add more

Table 2. M.S. Incidence Patterns: 1980-1990		
I.H.S. Area	Mean Annual Incidence By Reporting Area	Mean Annual Incidence Per 100,000
Aberdeen	1.3	1.7
Alaska	.1	.12
Albuquerque	.5	.8
Bemidji	.1	.12
Billings	.8	1.7
Navajo	.2	.10
Oklahoma	1.2	.5
Phoenix	.4	.4
Portland	.2	.16

information about the nature and inheritance of a resistance factor.

Another related factor to be considered is the variability of expression of the disease itself. It is possible that the native Indians have a "milder" form of the disease, similar to their genetically related Asian ancestors, which also may be the expression of a genetic resistance pattern.

Previous studies have also suggested a critical age factor of migration to and from different prevalence zones, as well as the age of certain childhood infections, as risk factors for later development of MS. Perhaps this age risk factor also exists as a result of "internal migration" within the high prevalence zones through the process of urbanization and relocation of certain tribes.

Preliminary data analysis indicates an incidence rate of MS in the Native American population of five- to ten-fold less than that of Caucasian populations in the same geographic areas. MS is essentially non-existent in the Amerindian population groups residing in the higher latitude regions where MS is highest for the Caucasians.

The mean annual incidence rate (MAI) over the ten-year period of this study indicates that the highest mean annual incidence rate of MS in Amerindians does indeed occur in the northern plains area where the Caucasian MS incidence and prevalence rates are also the highest. The MAI of the Sioux Nation area with a population base of 125,000 is 2.7/100,000.

The Southern Plains Amerindians in Oklahoma, with a population base of approximately 257,000, indicate a mean annual incidence rate of 1.2/100,000. This group was more closely studied and through (unidentified) chart review indicates a verification diagnostic rate of 80%.

Blood quantum analysis of this population group showed no patient with MS with more than 1/4 with a range of 5/256 to 1/4 and with a mean blood quantum of 1/8. The female to male ratio was 2:1 with an average age of onset of 34 years.

In the IHS reporting area of the Southwest U.S., the mean annual incidence rate varies (per 100,000) between: .1 (Navajo), .4 (Phoenix), and .8 (Albuquerque); an average annual incidence rate varying from one- to eight-fold in the same geographic area (Table 2).

The overall prevalence rate of MS in Caucasians in the U.S. is estimated to be 80/100,000 but with a significant geographic variance. It has not been established in the U.S. Native American population.

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a diffuse inflammatory disease of unknown etiology. Because it primarily involves the smaller blood vessels and connective tissue, lesions can affect all the organ systems of the body, including the central nervous system. Between 80% and 90% of the patients are female with an onset more common during young adulthood but occurring in all age groups. The disease may spontaneously terminate as an acute episode or may become remittent over a period of years or occasionally run a chronic progressive course, similar to MS.

Eighty to eighty-five percent of SLE patients will have articular disease sometime during their course, either polyarthalgias or a non-deforming migratory polyarthritis. Cutaneous nodules and myositis are less common. Systemic features of fatigue, fever, and weight loss occur in over 80% of SLE patients and 50% develop nephritis.

About one-third of the patients with SLE will develop neurological findings mimicking MS. These two diseases have many common clinical features as well as laboratory findings, and some investigators hypothesize that MS and SLE are both clinical manifestations of a similar autoimmune response. Familial clustering of these diseases and other diseases of immune dysfunction have also been noted.

HLA mapping studies have found significantly increased frequencies of HLA-DR2 DR3 and also the B cell alloantisera response defining MB-1 and MT-1, perhaps in association with hereditary deficiencies of the complement system, especially C2 and C4.[26,27]

In general there appears to be a higher incidence of SLE in Native American populations than previously reported but with significant disparity between regions and tribes. For example, the MAI per 100,000 ranges from 1.2 in the Albuquerque area to 4.1 in the Aberdeen area.

Morton et al.[28] reported in 1993 that the prevalence rates of SLE in Alaska of 92/100,000 in the Indian population as compared to 11/100,000 in the Eskimo (Inuit) population (Table 3).

Table 3. S.L.E. Incidence Patterns: 1980-1990

I.H.S. Area	Mean Annual Incidence By Reporting Area	Mean Annual Incidence Per 100,000
Aberdeen	3.6	4.1
Alaska	2.8	3.3
Albuquerque	.8	1.2
Bemidji	.8	1.3
Billings	1.6	3.2
Navajo	3.7	2.0
Oklahoma	3.9	1.5
Phoenix	3.2	3.0
Portland	1.7	1.4

Rheumatoid Disease

Rheumatoid arthritis affects approximately 1% of the adult population in the U.S. It may involve all joints but primarily the smaller joints of the hands and feet. Eight percent of patients with rheumatoid arthritis are positive for a rheumatoid factor, an autoantibody directed against immunoglobulin G.

Extra-articular disease may affect a variety of tissues including the skin, lungs, eyes, heart, and vascular system.

Seronegative spondyloarthropathies include ankylosing spondylitis, Reiter's syndrome, arthritis with inflammatory bowel disease, and psoriatic spondylitis. They are distinguishable by their different clinical patterns and also linked by their association with HLA-B27 tissue type.

Ankylosing spondylitis (AS) involves the axial-skeleton and is characterized by bony fusions. The etiology of AS is unknown, but the strong association with HLA-B27 suggests a genetic predisposition. The prevalence of AS is 1% to 2% of the population and more common in young adult men. The classic features of AS are chronic low back pain, fusion of the axial-skeleton with sacroiliitis, and involvement of the shoulders and hips. Extra-articular features include: pulmonary fibrosis, arthritis, heart block, and iridocyclitis.

Juvenile rheumatoid arthritis has an onset at less than sixteen years of age with a chronicity of greater than three months' duration. It is distinguishable by its pattern of presentation of the arthritis, consisting of polyarthritis (more than five joints involved), pauciarthritis (four or less joints), and systemic disease (Still's disease). Some children also appear to have a juvenile onset type of ankylosing spondylitis affecting mainly the large joints and lower extremities and sacroiliitis. Ninety percent of these children are HLA-B27 positive and share the clinical and immunogenetic features of the adult seronegative spondyloarthropathies.

Still's disease affects children usually less than five years of age and consists of polyarthritis, fever, rash, lymphadenopathy, hepatosplenomegaly, serositis, and elevated sedimentation rate leukocytosis.

ANA positive arthritis is also more common in younger children, characterized by large joint, lower extremity involvement with spontaneous remissions without deformity.

In 1969, Hill[29] and others drew attention to the increased frequency of chronic polyarticular arthritis in Canadian Indian children compared to non-Indian children and an increased prevalence of ankylosing spondylitis associated with the HLA-B27 antigen.

The seronegative spondyloarthriopathies are the most common cause of arthritis in the Alaskan Eskimo population while rheumatoid arthritis appears to be no more common than in the general population of the U.S. In sharp contrast, the Indians of southeast Alaska, who are predominantly Tlingit, are subject to high rates of rheumatoid arthritis like the Pima, Chippewa, and Yakima Indians of the U.S. Rheumatoid arthritis was not only more frequent but the peak age of incidence was also lower in this group.[30-34]

In general, a higher proportion of juvenile arthritis of the spondyloarthritic type was found in all the Alaska Indian and Eskimo children than that reported in predominantly white populations in the U.S., with an increased proportion of rheumatoid factor positive polyarticular disease in the Indian children.

A recently completed twenty-year longitudinal study conducted to estimate the incidence and preva-

Table 4. Rheumatoid Disease Incidence Patterns: 1980-1990		
I.H.S. Area	Mean Annual Incidence By Reporting Area	Mean Annual Incidence Per 100,000
Aberdeen	20.2	27
Alaska	19.5	23
Albuquerque	8.4	13
Bemidji	2.0	3
Billings	11.0	23
Navajo	17.2	9.5
Oklahoma	10.9	4.2
Phoenix	23.2	22
Portland	7.9	6.6

lence of rheumatoid arthritis in Southwestern American Indians in Arizona demonstrated an age adjusted incidence rate ten times higher than the Rochester study and six times higher than the Japan study, with an age-adjusted incidence rate of 42 per 10,000 person years. The female to male ratio of 2:1 was similar to that reported in the general population studies of rheumatoid arthritis in the U.S.[35]

The mean annual incidence rates from this ten-year study indicate another disparity by regional reports, varying from 3/100,000 (Bemidji) to 27/100,000 (Aberdeen), a nine-fold variance.

The highest incidence rates for rheumatoid diseases in the North American native populations apparently exist predominantly in the northern latitude areas of Aberdeen, Billings, and Alaska and then paradoxically in the southwestern reporting area of Phoenix (Table 4).

This occurrence of specific rheumatic diseases in excessive frequencies in some Native American populations suggests an unusual genetic susceptibility and/or environmental influence operating in these groups. That populations inhabiting such disparate environments as the Arizona desert and the Alaskan rain forest show such high susceptibility of RA supports the need for further genetic investigations.

The overall prevalence rate for RA in Caucasians in the U.S. is estimated to be 13,000/100,000 and unknown in the U.S. Native American population.

Summary

The data are too preliminary and incomplete to support any conclusionary statements. There is increasing evidence that the Native American populations are becoming more susceptible to developing the immunogenetic complex of diseases than previously reported. There are also a number of paradoxical tendencies observed, where certain anthropologically related native populations may have the lowest prevalence rate of one of the immunogenetic diseases and then the highest prevalence rate in another disease category. For example, the Athabascan-related native groups of Canadian and Alaskan Indians and the U.S. Navajos show a marked resistance to developing MS and a susceptibility to developing lupus.

Rheumatoid arthritis is a high prevalence disease in the North American Indian populations and not in the Eskimo population, while the seronegative spondyloarthropathies are the most common cause of arthritis in the Alaskan Eskimo. At the same time, a higher proportion of juvenile arthritis of the spondyloarthritic type exists in both the Alaska Indian and Eskimo children than in a comparable U.S. Caucasian population. Rheumatoid arthritis in some southwestern tribes in Arizona demonstrates an age-adjusted incidence rate ten times that of the general U.S. population.

There is obviously a need for further and more complete study of these immunogenetic disease processes as they relate to the Native American population. Such a study will involve a medical-ethnographic view as well as a socioeconomic (health care) vantage. Moreover, it will require a very sensitive and respectful approach to a very sensitive and respected culture—the Native Americans. J

*This study has been totally supported by Research to Prevent Blindness, Inc., New York City, New York.

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The Authors

Thomas E. Acers, MD, is Gaylord professor of ophthalmology at the University of Oklahoma Health Sciences Center in Oklahoma City and serves as a consultant for the Indian Health Service hospital in Claremore, Oklahoma.

Ann Acers-Warn, MD, is a graduate of Kansas University School of Medicine and currently is a resident physician in ophthalmology at the University of Oklahoma.

Post-Cholecystectomy Biliary Pain and Dyspepsia

(Response to 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors)

Hanna A. Saadah, MD

Background: The so called post-cholecystectomy syndrome is common, intractable, often progressive, causes prolonged suffering, and has no approved treatment. It usually presents with episodic biliary pains (colics), and postprandial dyspepsia (bloating and indigestion). Because treating a very recalcitrant case with lovastatin provided prolonged remission, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors were given to 12 subsequent patients with similar symptoms.

Objective: To determine whether HMG-CoA reductase inhibitors are useful in the therapy of post-cholecystectomy biliary pain and dyspepsia.

Methods: Open clinical trial in an internal medicine, private practice setting; data were collected from the patients' charts and from telephone interviews, five years after the index case had been treated.

Results: Eight of 12 patients experienced total resolution of their symptoms after many years of suffering; response occurred slowly within the first three months of treatment. Two other patients responded, stopped their medications, relapsed, and continue to be symptomatic. One patient did not take her medication and remains symptomatic; one other patient did not respond, was diagnosed with carcinoma of the pancreas,and died from it.

Conclusions: These preliminary results suggest that HMG-CoA reductase inhibitors may be useful in relieving the symptoms of this common and intractable disorder. Controlled studies are needed.

Direct correspondence to Hanna A. Saadah, MD, 4205 McAuley, Suite 400, Oklahoma City, OK 73120.

Cholecystectomy is commonly performed for the symptomatic relief of biliary pains and dyspepsia.[1,2] However, post-cholecystectomy surveys reveal that symptoms may persist in 23% to 47% of cases,[3] relapses tend to increase with time, [4] and currently, there are no accepted treatments for the post-cholecystectomy patients who continue to experience recurrent biliary symptoms.[4,5]

The following is a five-year observational study of 12 post-cholecystectomy patients who continued to have recurrent attacks of biliary pain and dyspepsia in spite of appropriate investigations and treatments, and who were finally treated with one of the three available 3-hydroxy-3-methylglutaryl coenzyme A-reductase inhibitors (HMG-CoA-RIs) namely: lovastatin, pravastatin, or simvastatin.

Index Case

In 1976, a 32-year-old woman underwent cholecystectomy for symptomatic cholecystolithiasis. Postoperatively, attacks of biliary pain and dyspepsia recurred with increasing intensity, causing frequent hospital admissions for the control of pain, nausea, vomiting, and dehydration. Three endoscopies with retrograde biliary cholangiograms were followed by exploratory laparotomy, cystic duct excision, and endoscopic sphincterotomy, all without relief. During the subsequent 10 years, the patient was hospitalized 4 to 5 times annually, each time for 5 to 10 days, and spent many days in bed (at home) with milder attacks. In 1986, a fourth endoscopic retrograde cholangiogram revealed a common bile duct stone which

Table 1. Ages and Symptom Durations in 12 Patients (4 Males & 8 Females)

	Average	Range	St. Dev.
Patients' Ages in Years	49	25 to 71	12.2
Age Symptoms Started	41	24 to 70	14.2
Symptom Duration in Years			
From: Start till CCMY	3.6	1 to 15	4.4
CCMY till Rx with HMG CoA RI	3.6	0.1 to 11	4.3
Start till Rx with HMG CoA RI	7.2	1 to 20	5.8

(HMG CoA RI = 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor; CCMY = Cholecystectomy; St Dev. = Standard Deviation; Rx = Treatment

was delivered endoscopically, but in spite of that, the patient's attacks continued to recur with the same frequency and intensity. Although she remained relatively asymptomatic between attacks, the frequent hospitalizations and absenteeism from work caused her to lose her job. Repeated attempts at medical therapy with low fat diet, cimetidine, chenodiol, antispasmodics, anti-reflux measures, and antacids failed to prevent the recurrent attacks.

In 1988, lovastatin (40 mg/day) was started with the hope of decreasing the cholesterol saturation of bile and thus inhibiting subsequent cholesterol crystallization and stone formation. By the end of the second month of lovastatin therapy, the patient had became totally asymptomatic and has remained well until the present time. She has continued to take her lovastatin faithfully for six consecutive years, has been re-employed, and remains free of biliary pains and dyspepsia.

Table 2. Symptoms in 12 Post-Cholecystectomy Patients

	Patients	%
Timing of Attacks		
Only nocturnal	4	33
Only post-prandial	3	25
Nocturnal & post-prandial	5	42
Duration of Attacks		
1-12 hours	6	50
1-7 days	6	50
Frequency of Attacks		
Daily	6	50
Weekly	3	25
Monthly	3	25
Symptom Complexes During Spells		
Nausea, bloating, belching, upper abdominal pains and burning, often referred to back-scapula-neck areas, with excessive sweating	12	100
Chest pains, palpitations, dyspnea	3	25
Diarrhea	2	17
Chills and Fever	1	8

Data and Results

Based on this clinical experience, 12 similar patients presenting with recurrent upper abdominal attacks of biliary pain and dyspepsia were treated with HMG-CoA-RIs and followed carefully in the office during the past 5 years. All of these patients had failed cholecystectomy and had numerous diagnostic work-ups including gastrointestinal barium studies and endoscopy, and all had failed repeated medical therapy for presumed peptic disease, reflux esophagitis, and irritable bowel syndrome. Symptom severity was variable, ranging from minor attacks of post-prandial bloating to severe biliary colics requiring narcotics and antiemetics. Tables 1 through 5 summarize the pertinent clinical features, findings, therapy, and outcomes of these 12 refractory patients.

Discussion

Cholecystectomy results in chronic desaturation of bile[6] and can relieve recurrent attacks of biliary pain and dyspepsia, even in the absence of cholecystolithiasis.[2] That these attacks of biliary pain and dyspepsia may be precipitated by the passage of cholesterol crystals or stones through the bile ducts is suggested by the fact that symptomatic common bile duct stones can occur after cholecystectomy[7] and can cause biliary pancreatitis.[8] Since common bile duct stones have been successfully dissolved with bile salts,[9] it is also possible that they can be dissolved with HMG-CoA-RIs. These agents desaturate bile by reducing cholesterol synthesis at the level of the hepatocyte. This, in turn, reduces the cholesterol concentration in bile which should favor an increase in cholesterol solubility with a decrease in cholesterol crystallization and subsequent stone formation.[10-13] Already, HMG-CoA-RIs in combination with ursodiol have been utilized successfully in the rapid dissolution of cholesterol gallbladder stones,[14] and are being evaluated for the therapy and prophylaxis of cholesterol gallstone disease.[15]

In this observational study, 8 of 12 post-cholecystectomy patients with refractory attacks of biliary pain and dyspepsia responded favorably to HMG-CoA-RIs. At the time of this writing, all eight patients con-

Table 3. Findings and Therapy in 12 Post-Cholecystectomy Patients	Patients	%
Hepato-Biliary Findings		
Positive Murphy's sign	6	50
Gallbladder stones	7	58
Gallbladder sludge &/or cholecystitis	5	42
Transaminase elevation < 2x normal	3	25
Transaminase elevation > 2x normal	1	8
Therapeutic Agents & Doses		
Lovastatin 20-40 mg/day	7	58
Pravastatin 20-40 mg/day	2	17
Pravastatin 40 mg + ursodiol 600 mg/day	1	8
Symvastatin 20 mg/day	1	8
Refused medication	1	8

Table 4. Lipid Values Before and During Treatment with HMG CoA RI in 12 Post-Chloecystectomy Patients	mg/dL	mmol/L	St. Dev.	% Decrease
Group Averages				
Initial cholesterol	206	5.3	39	—
Final cholesterol	164	4.2	28	20
Initial HDLC	46	1.2	10	—
Final HDLC	48	1.2	8	0
Initial LDLC	123	3.2	30	—
Final LDLC	91	2.4	24	27
Initial triglycerides	182	2.1	114	—
Final triglycerides	127	1.4	36	27

(LDLC = Low density lipoprotein chloesterol; HDLC = High density lipoprotein cholesterol)

tinue to take their medications and to feel well. In one of them, however, the HMG-CoA-RI alone provided only partial relief from symptoms, and had to be combined with ursodiol in order to achieve the desired freedom from attacks. Two other patients improved while on treatment but relapsed upon discontinuation of the HMG-CoA-RIs; these two remain intermittently symptomatic because of their personal inclinations to stay medication free.

In these selected patients, it is possible that persistent supersaturation of bile may have resulted in episodic passage of cholesterol crystals or stones during periods of high cholesterol synthesis. This would explain why biliary pains peak at midnight,[1] and why 9 of the 12 patients had nocturnal symptoms.

Hyperlipidemia was not a feature of this syndrome (Table 4), nor was the presence of gallbladder stones at cholecystectomy (Table 3). The literature clearly supports the notions that most gallbladder stones are silent, that symptoms commonly occur in the absence of gallbladder stones, and that cholecystectomy provides equal relief from symptoms for those with and those without gallbladder stones.[5] Therefore, it is possible that some of the biliary pains and dyspepsia so commonly associated with gallbladder stones may, in fact, be caused by cholesterol crystals or stones passing through the bile ducts.[16] This is certainly plausible because supersaturation of bile may be episodic, while both cholecystolithiasis and cholecystis are usually chronic and persistent. Indeed, cholecystectomy may not only relieve symptoms by removing the sick gallbladder, but also by chronically desaturating bile, a consequence similar to that afforded by the HMG-CoA-RIs, although via a different mechanism.[6] Therefore, when cholecystectomy fails to provide the desired relief, additional therapy with HMG-CoA-Rs should, theoretically, lead to even greater desaturation of bile and better protection from recurrent attacks of biliary pain and dyspepsia. This concept is supported by the results obtained from this small open study. However, because the so called post-cholecystectomy syndrome is common,[3,4] may cause chronic morbidity, and has no accepted therapy, it is essential that these preliminary results be confirmed in controlled trials, as they might have wide clinical applications.

Table 5. Durations of Therapy, Response Times, and Outcomes in 12 Post-Cholecystectomy Patients	Average	Median	Range	St. Dev.
Group Times in Months				
Duration of HMG CoA RI Rx at end of study	26	12.5	2 to 62	25
Time of optimal response while on Rx	2	2	1 to 6	1.4
	Patients	**%**		
Final Outcome on Rx				
Completely well	8	67		
Improved, stopped, relapsed	2	17		
Refused Rx, remains symptomatic	1	8		
No response, died of pancreatic ca.	1	8		

In the meantime, refractory post-cholecystectomy patients who continue to have recurrent attacks of biliary pain and dyspepsia in spite of appropriate diagnostic work-ups and medical therapy for peptic disease, reflux esophagitis, or irritable bowl syndrome should be offered a two-month therapeutic trial with HMG-CoA-RIs. If they should respond, it would then be reasonable to place them on maintenance therapy because the benefits of HMG-CoA-RIs in this population of patients seem to exceed the small potential risks.

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The Author

Hanna A. Saadah, MD, FACP, is a clinical professor of medicine at the University of Oklahoma Health Sciences Center in Oklahoma City.

Current and Future Uses of Biological Response Modifiers in the Treatment of Viral Hepatitis

David H. Van Thiel, MD

The author reviews the use of interferon α and/or additional cytokines for the treatment of chronic viral hepatitis B and hepatitis C.

Biological response modifiers (BRM) are small molecular weight substances typically being glycoproteins having a molecular weight between 15 and 45 kD that are produced by cells and effect the activation state of other cells and occasionally the cell of their own origin. Another commonly used name for BRM is cytokines.

The use of BRM for the treatment of viral hepatitis is less than 20 years old. It began with and the longest experience is with interferon α (IFN) More recently, other BRM agents have been used for this purpose as well.

Because the experience is greatest with IFN, much of what follows will be a review of the use of IFN for the treatment of chronic viral hepatitis, both hepatitis B (HBV) and hepatitis C (HCV). This will be followed by a presentation of the use of additional cytokines used either alone or in combination with IFN to treat chronic HBV or HCV liver disease.

Hepatitis B (HBV)

Interferon.— Rather than review all of the reports available on the use of IFN for the treatment of HBV disease, it is simpler to review a recent meta-analysis of 15 randomized controlled studies in which 837 patients with chronic hepatitis B were analyzed.[1] In this meta-analysis, the only studies used were those where the IFN treatment was given for at least 3 months and an additional 6 months of follow-up were included. Only 7.8% of the cases treated became HBsAg negative. Although small, this was four-fold greater than that seen in the placebo-treated control groups ($p \leq 0.001$). Thirty-three percent of the IFN-treated subjects became eAg negative as compared to 12% of the placebo treated controls ($p \leq 0.0001$). Most importantly, 37% of the IFN-treated cases and only 17% of the placebo-treated controls became HBV-DNA negative ($p \leq 0.0001$).

Based upon these results, it was concluded that IFN is effective at terminating HBV replication in a third of treated cases. The authors of this analysis worried, however, that further follow-up was required to determine whether this benefit would relate to a reduced rate of cirrhosis or hepatocellular carcinoma in IFN-responding patients. This warning is appropriate as it is quite clear that, at least in some cases, the disruption of the hepatic anatomy is sufficient prior to the time that an IFN response is achieved, that despite loss of viral replication and HBsAg, the microvascular disease has progressed to a point that it will continue to progress to cirrhosis despite the absence of a persistent viral infection. Since hepatocellular carcinoma (HCC) is thought to be a consequence of chronic inflammation rather than a direct result of the HBV infection per se, it is highly likely that once the process of HCC development is initiated by the virus, it can progress as a consequence of continuing chronic inflammation even in the absence of persistent viral infection.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

The clinical and serologic long-term consequences of an IFN response in a patient with HBV-associated liver disease has been reported by investigators at the NIH.[2] Specifically, these investigators identified 23 patients, who had been the responders in one or another of their various IFN protocols. In each case, a response was defined as a loss of HBV-DNA, HBV-DNA-polymerase, HBeAg, and an ALT level less than or equal to a doubling of the upper limit of normal. These 23 cases represented 32% of the entire population with HBV disease they treated. As a group they had been followed for a mean of 4.5 years. Eighty-seven percent of these IFN responders continued to be HBeAg negative and HBV-DNA negative during the follow-up period. Fifty-seven percent eventually became HBsAg negative. Thirteen percent experienced a disease relapse in the first year of follow-up. An additional patient relapsed after 6 years of follow-up.

In response to IFN treatment, essentially all responders became DNA-polymerase negative during the course of IFN therapy. Sixty percent became HBeAg negative during the course of their IFN therapy. All became eAg negative during the 6 months of follow-up after discontinuing the IFN therapy. The ALT level became normal in 80% of the responders but lagged well behind the conversion to eAg negative with only 70% having normal ALT levels at 1 year and only an additional 10% achieving normal ALT levels over a subsequent 3-year period of continued observation. Most interesting was the finding that a seroconversion to HBsAg negative and loss of PCR-detectable HBV-DNA occurred over the 1- to 4-year period after the IFN had been discontinued.

The authors of this report concluded that remissions of HBV disease in IFN-treated patients, who are HBV-DNA and eAg positive at entry, are long-lasting in the majority of cases. Moreover, most responders loose HBsAg over a period of several years of follow-up. They noted that this pattern is similar to that seen in cases that spontaneously remit.

Thymosin.— The newest agent on the therapeutic horizon for treatment of HBV disease is thymosin $\alpha 1$.[3,4] An advantage of thymosin $\alpha 1$ as compared to IFN is that a reproducible ELISA assay exists for thymosin $\alpha 1$, which enables thymosin $\alpha 1$ levels to be measured either in serum or urine. Moreover, the assay has enabled pharmacokinetic studies to be performed. Specifically, it has been shown that repetitive administration of thymosin $\alpha 1$ to subjects over a four-fold dose range, encompassing the anticipated therapeutic dosing schedule, produces reproducible peak levels that vary as a function of dose. Thymosin $\alpha 1$ administered subcutaneously has a T $^1/_2$ of 1.49 to 2.61 hours.

Possibly of more importance clinically is the observation that repetitive administration of thymosin $\alpha 1$ is not followed by a depression of the WBC count as occurs with IFN.[3,4] Thymosin $\alpha 1$ administration also produces a significant ($p \leq 0.05$) increase in the number of circulating natural killer cells present in peripheral blood.

The initial clinical experience with thymosin $\alpha 1$ in humans was reported by Mutchnick et al.[5] In this study, a total of 12 subjects, who were seropositive for HBV-DNA and HBsAg and had 6 months or more of biochemical treatment as well as histologic liver disease, were randomized to receive either thymosin (900 $\mu g/m^2$ 2 $\times$ w), thymosin fraction V (900 $\mu g/M^2$ 2 $\times$ w) or placebo for 6 months. After 6 months of post-treatment follow-up, 14% of the active treatment groups were HBV-DNA positive while 80% were still positive in the placebo-treated group ($p \leq 0.04$). In terms of the rate of seroconversion from eAg to eAb positive, 86% of the thymosin-treated subjects achieved this goal, while only 40% of the placebo-treated controls did. Most importantly, only 14% of the thymosin $\alpha 1$ treated subjects had immunohistochemical-demonstrable HBcAg in their liver after 6 months of follow-up, while 80% of the controls did ($p \leq 0.04$). Even more important may be the observation that after a mean follow-up of 27±3 months that 86% of the thymosin-treated responders continued to be in remission.

In terms of side effects, none were experienced with thymosin treatment, while some of the subjects treated with thymosin fraction V experienced pain at their injection sites.

These promising results with thymosin $\alpha 1$ were extended in an additional 20 patients who were HBV-DNA and HBsAg positive with chronic liver disease.[6] Specifically, 75% of the thymosin $\alpha 1$-treated patients cleared HBV-DNA from their serum, while only 25% of the controls did. Seventy-eight percent of the thymosin responders have remained in remission for periods of up to 5 years. Importantly, all long-term responders have normal ALT levels.

Recently, an Italian group of investigators has compared the efficacy of IFN vs thymosin $\alpha 1$ in a direct comparison trial of 14 patients who had chronic active hepatitis due to a precore mutant form of the HBV.[7] The thymosin $\alpha 1$-treated group received 900 $\mu g/m^2$ of thymosin 2 $\times$ w $\times$ 26 weeks, while the IFN-treated group received 5 MU IFN 3 $\times$ week $\times$ 26 weeks. Twenty-nine percent of the thymosin $\alpha 1$- and none of the IFN-

treated group achieved normal ALT levels. Seventy-one percent of the thymosin α1-treated subjects and 43% of the IFN-treated subjects became HBV-DNA negative at the end of the 26-week treatment period. These data suggest that thymosin α1 may be more effective in the treatment of the chronic hepatitis due to the HBV virus than is IFN.

IL-2.— Interleukin-2 (IL-2) has also been used to treat cases of chronic active hepatitis due to the HBV.[8] The doses of IL-2 used in the initial reported study ranged from 0 to 3.6 MIU IL-2 administered SQ daily for 56 days (8 weeks). In this initial trial, IL-2 failed to be better than no treatment at eradicating HBV-DNA. Worse yet, the use of IL-2 was associated with considerable side effects. Thus, when used alone, IL-2 does not appear to be useful in the treatment of HBV-associated chronic liver disease.

Bauch et al have utilized a combination of IFN and IL-2 vs IFN alone in a randomized controlled prospective trial of 37 patients with histologically confirmed chronic hepatitis B who were HBV-DNA and HBsAg positive.[9] The group that received IFN + IL-2 were given 3 MU IFN SQ 3 × week for 5 months, 1.5 CU IL-2 the first 2 days of each month beginning with the second month of treatment, and 0.3 CU IL-2 5 days/wk for the remainder of each month. The group that received only IFN was given a slightly higher dose of IFN, 5 MU 3 × w × 5 months. The response of both groups was poor, being 24% and 28% for clearance of HBV-DNA, eAg, and ALT normalization. The side effects of treatment were severe in the group receiving IL-2 in combination with IFN. The authors of this study concluded that monotherapy with IFN is preferable to the combination of IFN + IL-2 for the treatment of chronic hepatitis B liver disease.

TNFα.— A group of Japanese investigators have examined the effect of yet another BRM TNFα alone and in combination with IFNγ.[10] Because of the recognized toxicity of TNFα, these investigators initiated their studies using a HBV-producing cell line (HB611 cells) instead of humans as their experimental model. To 12d cultures they added TNFα 0-1000 μ/ml alone or in combination with 0-1000 μ/ml IFNγ and examined the effect of such treatment on HBV-DNA production by the cell line. Both agents produced a dose-dependent inhibition of HBV-DNA production to a maximum of 55% for TNFα and 51% for IFNγ. A maximal inhibition of 72% was achieved with maximal doses of both agents used together. Neither cytokine was able to eliminate HBV-DNA or HBeAg production by the model cell line. The authors of this report rightly concluded that the two cytokines had a synergetic effect upon HBV replication. However, they chose not to pursue their investigations further in humans because of the toxicity of TNFα and the poor efficacy seen in their in vitro model system.

GM-CSF.— GM-CSF, or granulocyte macrophage colony stimulating factor, is yet another cytokine which has been used to treat patients with chronic active hepatitis due to the HBV.[11] In a pilot study, 9 patients with chronic HBV liver disease were given 0, 0.4, 2, or 3 μg/kg GM-CSF alone or in combination with IFN 5 MU daily. All GM-CSF-treated subjects experienced a reduction in their HBV-DNA level. Half of those who completed the study became HBV-DNA and eAg negative and experienced an improvement in their histopathology. Two patients actually obtained normal ALT levels. As expected, in all cases receiving GM-CSF, a dose-dependent increase in WBC counts was observed. Those who received both GM-CSF and IFN had a more pronounced reduction in their HBV-DNA level.

Hepatitis C (HCV)

In contrast what is known about how to use IFN to achieve the best results, much more has been accomplished in cases of HCV disease than has been the case with HBV-related disease. Both of the two original studies reported utilizing IFN to treat HCV disease utilized two different doses of IFN.[12] In the larger study, 166 adults with chronic non-A, non-B hepatitis (NANB) were given either no treatment, 1 MU, or 3 MU IFN SQ 3 × w for 6 months. When the results of the higher dose of IFN were compared to the placebo, 38% vs 4% experienced a normalization of their ALT level, while an additional 7% as compared to 4% experienced a ≥50% reduction in their ALT level. Unfortunately, 51% of the responding patients relapsed when the IFN was discontinued. Nonetheless, the authors of this paper were able to conclude that IFN was effective therapy for controlling disease activity in patients with chronic hepatitis of the NANB (HCV) type.

More recently, Rakela and associates, in an open label randomized parallel dose study, used 1, 3, or 6 MU IFN 3 × w for 24 weeks in 336 patients with chronic hepatitis C characterized by an elevated ALT and abnormal liver biopsy.[13] The response rate achieved increased with the dose of IFN used from a rate 17% with 1 MU to a rate of 46% with 3 MU and 62% with 6 MU 3 × w. More importantly, the sustained complete response rate increased from 4% to 7% and 15% as the dose of IFN was increased respectively. The authors of this study concluded that 6 MU 3 × w was more effective than was either 1 or 3 MU and was equally well tolerated by patients.

Japanese investigators have utilized even larger doses of IFN.[14] Moreover, they have developed the concept of high dose induction therapy. Specifically, Iino and associates[14] treated three groups of patients with a total dose of 480 MU of IFN administered as follows: Group 1 received 10 MU 6 days per week × 8 weeks; Group 2 received 10 MU 6 days per week × 4 weeks, followed by 8 weeks of 3 × week; Group 3 received 2 weeks of 10 MU 6 days per week, followed by 12 weeks of 10 MU 3 × week. The response rate for Group 1 was 21.9%; for Group 2 it was 42.3%; and for Group 3 it was 51.5%. Based upon these results, the researchers concluded that an induction course of high dose intensive IFN therapy followed by a lower dose albeit higher than those administered in the West was more successful than continued high dose therapy.

A further twist on how to use IFN in cases of HCV chronic hepatitis was developed by a group in Italy.[15] These investigators treated 90 patients divided into two groups. The first group received 3 MU 3 × w for 12 months. The second group received 3 MU 3 × w for 10 months, followed by 1 month of 2 MU 3 × w and a second month of 1.5 MU 3 × w. Thus, the second group actually received less total IFN therapy but obtained an equal number of complete responses. What was of interest was the fact that the second group had a significantly greater number ($p \leq 0.01$) of sustained responses, such that 48% of the first group that received continuous high dose therapy relapsed while only 14% of those who received a terminal tapered dose relapsed.

Based upon these data and the data of Iino and associates, it would appear as if the ideal IFN regimen for HCV disease would be high dose induction therapy (possibly 10 MU daily × 3 wks), followed by 6 to 11 months of moderate therapy (3-5 MU daily), and then a final 2-month taper during which 2 MU and then 1.5 MU is used 3 × w, each for a month.

The most recent addition to the armamentarium used to treat HCV disease has been the addition of phlebotomy therapy to IFN in an effort to reduce the rate of HCV replication to a level where IFN therapy has the potential of terminating viral replication. This concept was developed from the observation that patients who responded to IFN at standard doses had lower hepatic iron levels than did those who failed to respond.[16]

With this observation, two groups[17,18] have treated patients who previously failed to respond to IFN by combining iron reduction therapy, consisting of repetitive phlebotomy therapy, with their standard IFN protocols. With this combination of therapies both groups have noted that individuals who previously had failed to respond to IFN therapy, now did. Importantly, one of these two groups has also noted that those who respond to the combination of IFN plus iron reduction therapy rarely relapse as compared to what is seen with most successful IFN alone regimens.

Conclusions

It is quite clear from the preceding that hepatologists are still learning how to use IFN to treat viral hepatitis. It is also quite clear that additional agents are being developed to treat viral hepatitis and that some of these show promise as anti-viral agents either alone (thymosin α1) or in combination with IFN (GM-CSF and iron reduction therapy). Clearly the time for active therapy of viral hepatitis has arrived. The future, both for hepatologists and their patents, appears to be most promising.

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The Author

David H. Van Thiel, MD, is director of the liver disease section and medical director of transplantation at the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City.

SPECIAL

Medical Leases and Patient Referrals Under Oklahoma Law: Are They Mutually Exclusive?

T.P. Howell, JD

This article discusses the impact of Oklahoma's new patient referral law on leases between medical care providers. The statute appears to ban certain practices that could be quite common, so the article offers some advice on how to avoid running afoul of the statute.

In recent years, the health care industry has been rapidly consolidating. Employers and insurance companies have encouraged the formation of health maintenance organizations and preferred provider networks, and hospitals have broadened their income base through acquisitions and joint ventures. At the same time this consolidation has been proceeding, however, and perhaps to some extent as a consequence of it, federal and state governments have increased their regulation of the industry. For instance, on May 10, 1993, the Oklahoma legislature enacted House Bill No. 1719, which prohibits certain types of patient referrals.

Numbered as Section 1-742 of Title 63, the statute provides that any person who "pays to or accepts *anything of value* from" any entity "*for securing or soliciting patients* for any hospital or other entity providing health care services in this state" shall be guilty of a misdemeanor. The statute further provides that a violation shall be grounds for disciplinary action by the state agency which licenses the offender.[1]

On its face, this statute is very broad. If *anything* of value is given to *anyone* in return for a patient referral, both parties to the transaction are subject to prosecution and loss of their licenses. The statute is not limited to referrals of patients covered by Medicare or Medicaid, but applies to *all* patient referrals. Whether the apparent breadth of this statute can be reconciled with the reality of current medical practice is an important question for both physicians and hospitals.

For instance, many hospitals in Oklahoma and elsewhere in the country are expanding their operations beyond their primary locations. They are establishing or becoming associated with laboratories, physical and occupational therapy clinics, diagnostic radiology and radiation therapy facilities, cardiac monitoring units, home health care services and other outpatient care facilities.[2] Such a facility often leases space from a hospital, or conversely a unit of a hospital sometimes leases space in premises owned by the facility. Under such an arrangement, the parties might be expected to make referrals to each other. Are such referrals permitted under Oklahoma law?

To date, there have been no published decisions which interpret the new Oklahoma statute. That statute, however, appears to be based upon the so-called anti-kickback provisions in the Medicare and Medicaid statutes, enacted in 1972 and amended several times since. A number of cases construing those provisions have discussed the definition of a "kickback."[3] More importantly for the purposes of this paper, one case has discussed whether the lease of premises by a physician to an entity which provides medical-re-

Direct correspondence to T.P. Howell, 2900 Oklahoma Tower, 210 Park Avenue, Oklahoma City, OK 73102-5605.

lated services to the physician's patients can constitute an illegal arrangement.

In that case,[4] a general practitioner bought the building in which he maintained his medical practice. A hospital had operated a pharmacy in that building, and after the purchase, the physician, Dr. Tanquilut, asked the employee pharmacist to continue to operate the pharmacy as its owner. The base rental for the pharmacy was to be $4,000.00 per month, together with "incentive rent" based on the pharmacy's operating costs. About four and one-half years later, Dr. Tanquilut was notified by the Illinois Department of Public Aid that he could no longer participate in its Medical Assistance Program (which administered Illinois's Medicaid program) because he had allegedly violated federal law by receiving kickbacks and rebates.[5]

After several rounds of hearings, the Appellate Court of Illinois reviewed the evidence which had led to Dr. Tanquilut's termination from the Medical Assistance Program. He had been charged with violating the statute which provides for the felony conviction of anyone who "knowingly and willfully solicits or receives any remuneration (including any kickback, bribe or rebate) directly or indirectly, overtly or covertly, in cash or in kind, in return for referring an individual" to a provider whose services are paid for by Medicaid.[6] The remuneration the doctor was charged with receiving was excess rent. The Appellate Court pointed out, however, that the department had not introduced *any* evidence of comparative rents in the area where the pharmacy was located. Hence there was no support for the department's conclusion that the rent received by the doctor was excessive.[7]

Without any evidence of excessive rent, the court noted that the charges could be proved in only two other ways. "Either there must be evidence of patient steering or excess prescription practices by the doctor in return for the lease agreement, or the lease itself must be so open a violation as to invoke some sort of per se rule, perhaps because of the percentage arrangement, against its very existence."[8]

The court found that there was no evidence of "patient steering" or excessive prescription practices by the doctor. With regard to the second possibility, the court found that the lease was not a per se violation of the statute because the incentive rent was not a pure percentage rent arrangement (which had been discussed as a common abuse at the Congressional hearings on the statute).[9] Dr. Tanquilut was exonerated and his termination from Illinois' Medical Assistance Program was reversed.[10]

The Medicaid statute at issue in the *Tanquilut* case and Oklahoma's anti-kickback statute are quite similar. The Oklahoma statute forbids the payment or receipt of *anything of value* in return for securing or soliciting patients.[11] The federal statute bars the receipt of any *remuneration* in return for referring an individual to a medical service provider.[12] "Remuneration" has been interpreted very broadly.[13] Consequently, transactions permissible under the federal statute should also be permissible under the Oklahoma statute. The Oklahoma statute makes this presumption explicit by stating that the statute "shall not be construed to prohibit any payment, business arrangements or payments practice not prohibited by 42 U.S.C. §1320a-7b(b),"[14] the Medicaid anti-kickback statute discussed in the *Tanquilut* case.

Under Oklahoma law, then, and the precedent set in the *Tanquilut* case, a lease arrangement between a doctor and another provider of medical services or products should not be a violation of the anti-kickback statute if:

1. The rent is comparable to other rents in the area for similar facilities;
2. The lease does not provide any compensation, direct or indirect, for patient referrals (which specifically excludes percentage payments based on gross or net income of the lessee); and
3. The lease does not provide for "patient steering" by either party.

This interpretation was recently confirmed by the federal Department of Health and Human Services. On July 29, 1991, DHHS promulgated certain "safe harbor" regulations.[15] Those regulations exempt from prosecution or civil sanctions certain practices that might otherwise appear to be violations of the federal anti-kickback statutes. One of those exemptions applies to rent payments between providers of medical services. In order for a lease to fall within the safe harbor, it must be a written lease with at least a one-year duration. The rent must be based upon the fair market value of the leased premises and cannot take into account any referrals or other business generated between the parties.[16] These requirements are basically the same as those set forth in the *Tanquilut* case.

The next question is, assuming that the lease itself does not violate the statute and there is no "side arrangement" otherwise providing for compensation for referrals, may medical service providers in a landlord-and-tenant relationship make referrals to each other? Under those conditions, the referral is legal as long as the provider making the referral does not

have an ownership interest in the other. A provider who makes a referral to a facility in which she or he has an ownership interest, however small, logically would receive something "of value" from the referral, because it would tend to increase the value of the provider's investment.

This reasoning is confirmed by a Washington state case, *Day v. Empire Optical, Inc.*.[17] There, five ophthalmologists in partnership rented their office from a corporation they owned, Inland Empire Optical Company. Their offices were on the second floor of the building. On the first floor of the building, the optical company sold glasses. The doctors were sued for violating a Washington statute which prohibited the receipt of compensation for referrals of patients.[18]

The Washington Supreme Court noted that the ophthalmologists had a sign posted in their office which stated "we have an optical shop downstairs for your convenience," although the sign also said "please feel free to take your prescription to the optician of your choice." Still, eighty to ninety percent of the doctors' patients had their eyeglass prescriptions filled by the company downstairs.[19]

Inevitably, the doctors were benefitted when their patients bought eyeglasses from their company.

> The accumulation of profits, increase in value of the common stock, growth in net worth, possible tax advantages, and right to a distribution of income from Inland Empire Optical accruing to the defendant doctors thus amounted to the receiving by them directly or indirectly of a rebate, refund, commission, unearned discount or profit, or other valuable consideration in connection with the referral of their patients.[20]

Hence, because of the benefits the doctors received from their referrals, the court ordered that the doctors either divest themselves of their ownership of the optical company, or alternatively "neither directly or indirectly either verbally or in writing [*sic*], or by sign, symbol or gesture, or by physical arrangement of their offices, refer their patients to the optical company, or indicate in any way their hopes that the patient take his prescription there."[21]

The *Day* case stands for the proposition that a medical service provider who refers a patient to the provider's landlord or tenant receives value if the provider has an ownership interest in the landlord or tenant. Thus, such a referral would be illegal under Oklahoma law. (In many cases, federal law would also prohibit such referrals, but Oklahoma law is broader because it covers *all* providers, not just those who are reimbursed by Medicare or Medicaid.)

The bottom line of this analysis is that medical service providers can lease space from each other, but referrals cannot form any part of the compensation, and the referring party cannot have any ownership interest in the other. As long as the parties to a lease agreement have no other relationship with each other, however, and it is a bona fide, arm's length lease, they should be able to make referrals to each other. Of course, any such referral should be based upon the best interests of the patient, but certainly convenience for the patient is a factor to be considered.

Because of the relative dearth of explicit legal authority in Oklahoma on the subject, physicians and health care facilities might be wise to incorporate a referral policy in their lease agreements. The lease should state that neither party has entered into the lease on the expectation of receiving referrals from the other; that neither party will receive anything of value in return for making a referral; and that any referrals which are made will be based solely upon the best interests of the patient, including quality of service and convenience. Such language would not guarantee immunity from any problems, but it could tend to ameliorate the consequences of an investigation. Under federal law, "there is little likelihood of prosecution in situations where entities have sought legal advice and are making a good faith effort to comply with the safe harbor standards."[22] The same concept would probably apply to proceedings under Oklahoma's patient referral statute. ❑

References

1. The full text of the statute is as follows:
   - A. 1. Any person who intentionally or knowingly from any person, firm, association of persons, partnership or corporation for securing or soliciting patients for any hospital or other entity providing health care services in this state, upon conviction, shall be guilty of a misdemeanor and shall be punished by a fine of not less than Five Hundred Dollars ($500.00) and not more than Two Thousand Dollars ($2,000.00).
     - 2. In addition to any other penalties or remedies provided by law
       - a. a violation of this section by a health care professional or health care provider shall be grounds for disciplinary action by the state agency licensing, certifying or registering such professional or provider, and
       - b. the state agency licensing, certifying or registering such professional or provider may institute an action to enjoin violation or potential violation of this section. The action for an injunction shall be in addition to any other action, proceeding or remedy authorized by law.
   - B. This section shall not be construed to prohibit.
     - 1. Advertising, except that advertising which:
       - a. is false, misleading or deceptive,
       - b. advertises professional superiority or the performance of a professional service in a superior manner, and
       - c. is not readily subject to verification;
     - 2 2. Remuneration for advertising, marketing or other services that are provided for the purpose of securing or soliciting patients, provided the remuneration is:
       - a. set in advance,
       - b. consistent with the fair market value of the services, and
       - c. not based on the volume or value of any patient referrals or business otherwise generated between the parties; and
     - 3. Any payment, business arrangements or payments practice not prohibited by 42 U.S.C., Section 1320a-7b(b), or any regulations promulgated pursuant thereto.

C. This section shall not apply to licensed insurers, including but not limited to, group hospital service corporations or health maintenance organizations which reimburse, provide, offer to provide or administer hospital, medical, dental or other health-related benefits under a health benefits plan for which it is the payor when it is providing those services under a health benefits plan.

D. For purposes of this section:

1. "Health care professional" means any person who offers or provides counseling or health or mental health care under a license, certification or registration issued pursuant to Title 59 of the Oklahoma Statutes; and
2. "Health care provider" means any hospital or related institution offering or providing health care services licensed pursuant to Section 1-702 of this title.

2. Kornreich ES, Fields GI,Scharf SF. Amendments to Stark Laws curb referrals, *National L.J.*, October 11, 1993, at 25.
3. See, e.g., United States v. Duz-Mor Diagnostic Laboratory Inc. 650 F.2d 223 (9th Cir 1981); United States v. Hancock 604 F.2d 999 (7th Cir.), *cert. denied*, 444 U.S. 991 (1979); United States v. Zacher, 586 F.2d 912 (2nd Cir. 1978).
4. *Tanqutlut v. Illinois Department of Public Aid*, 396 N.E.2d 1126 (Ill. App. Ct. 1979).
5. *Id.* at 1128.
6. 42 U.S.C. §1320a-7b(A) (1990).
7. 396 N.E.2d at 1131.
8. *Id.*
9. 1977 U.S.C.C.A.N. 3039 *et seq.*
10. *Id.* at 1132
11. Okla. Stat. tit. 63, §1-742(A)(1993).
12. 42 U.S.C. §1320a-7b(b) (1990).
13. *United States v. Greber*, 760 F.2d 68, 71-72 (3rd Cir.), *cert. denied*, 474 U.S. 988 (1985).
14. Okla. Stat. tit. 63, §1-742(B)(3) (1993).
15. 42 C.F.R. §1001.952 (1991).
16. 42 C.F.R. §1001 952(b) (1991).
17. 456 P.2d 1011 (1969).
18. *Id.* at 1013.
19. *Id.* at 1015.
20. *Id.* at 1018.
21. *Id.* at 1019.
22. Tedricks S. Legal issues in physician self-referral and other health care business relationships, 13 *J. Legal Med.* 521, 541 (1992).

The Author

Mr. Howell is a director of the Oklahoma City law firm of Day, Edwards, Federman, Propester & Christensen.

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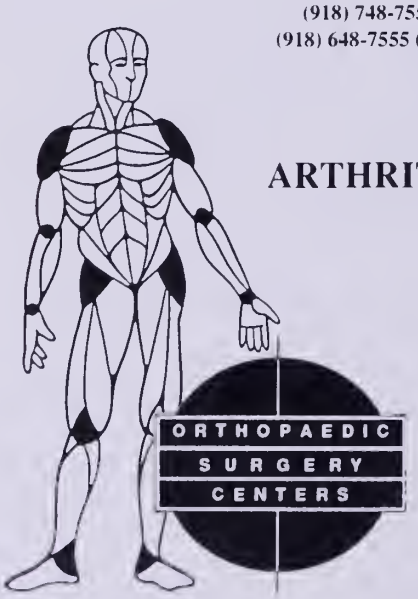

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Thou shall not...

State licensing board adopts new unprofessional conduct rules

The Oklahoma Board of Medical Licensure and Supervision adopted amendments to the unprofessional conduct rules in February, reports the spring 1994 issue of *Issues and Answers*, the board's quarterly newsletter. The changes were adopted on an emergency basis and became effective April 4, 1994.

The remainder of the board's 1994 meeting schedule appears in THE LAST WORD.

Following is the text of the section on unprofessional conduct as it now reads:

435:10-7-4. Unprofessional conduct

The Board has the authority to refuse, revoke, or take other disciplinary action against a licensee or certificate holder for unprofessional conduct. Pursuant to 59 O.S., 1991, Section 509, "Unprofessional Conduct" shall be considered to include:

(1) Indiscriminate or excessive prescribing, dispensing or administering of Controlled or Narcotic drugs.

(2) Prescribing, dispensing or administering of Controlled substances or Narcotic drugs in excess of the amount considered good medical practice or prescribing, dispensing or administering controlled substances or narcotic drugs without medical need in accordance with published standards.

(3) The habitual or excessive use of any drug which impairs the ability to practice medicine with reasonable skill and safety to the patient.

(4) Issuing prescriptions for Narcotic or Controlled drugs to minors in violation of 63 O.S. 1978 Supp., Sections 2601 through 2606, as amended.

(5) Purchasing or prescribing any regulated substance in Schedule I through V, as defined by the Uniform Controlled Dangerous Substances Act, for the physician's personal use.

(6) Dispensing, prescribing or administering a Controlled substance or Narcotic drug without medical need.

(7) The delegation of authority to another person for the signing of prescriptions for either controlled or non-controlled drugs.

(8) Fraud or misrepresentation in applying for or procuring a medical license or in connection with applying for or procuring periodic reregistration of a medical license.

(9) Cheating on or attempting to subvert the medical licensing examination(s).

(10) The conviction of a felony or any offense involving moral turpitude whether or not related to the practice of medicine and surgery.

(11) Conduct likely to deceive, defraud, or harm the public.

(12) Making a false or misleading statement regarding skill or the efficacy or value of the medicine, treatment, or remedy prescribed by a physician or at a physician's direction in the treatment of any disease or other condition of the body or mind.

(13) Representing to a patient that an incurable condition, sickness, disease, or injury can be cured.

(14) Willfully or negligently violating the confidentiality between physician and patient to the detriment of a patient except as required by law.

(15) Gross or repeated negligence in the practice of medicine and surgery.

(16) Being found mentally incompetent or insane by any court of competent jurisdiction; commitment to an institution for the insane shall be considered prima facie evidence of insanity of any physician or surgeon.

(17) Being physically or mentally unable to practice medicine and surgery with reasonable skill and safety.

(18) Practice or other behavior that demonstrates an incapacity or incompetence to practice medicine and surgery.

(19) The use of any false, fraudulent, or deceptive statement in any document connected with the practice of medicine and surgery.

(20) Practicing medicine and surgery under a false or assumed name.

(21) Aiding or abetting the practice of medicine and surgery by an unlicensed, incompetent, or impaired person.

(22) Allowing another person or organization to use a physician's license to practice medicine and surgery.

(23) Commission of any act of sexual abuse, misconduct, or exploitation related or unrelated to the licensee's practice of medicine and surgery.

(24) Prescribing, selling, administering, distributing, ordering, or giving any drug legally classified as a controlled substance or recognized as an addictive or dangerous drug for other than medically accepted therapeutic purposes.

(25) Except as otherwise permitted by law, prescribing, selling, administering, distributing, ordering, or giving to a habitue or addict or any person previously drug dependent, any drug legally classified as a controlled substance or recognized as an addictive or dangerous drug.

(continued)

Unprofessional *(continued)*

(26) Prescribing, selling, administering, distributing, ordering, or giving any drug legally classified as a controlled substance or recognized as an addictive or dangerous drug to a family member or to himself or herself.

(27) Violating any state or federal law or regulation relating to controlled substances.

(28) Obtaining any fee by fraud, deceit, or misrepresentation, including fees from Medicare, Medicaid, or insurance.

(29) Employing abusive billing practices.

(30) Directly or indirectly giving or receiving any fee, commission, rebate, or other compensation for professional services not actually and personally rendered, though this prohibition shall not prohibit the legal function of lawful professional partnerships, corporations or associations.

(31) Disciplinary action of another state or jurisdiction against a or license or other authorization to practice medicine and surgery based upon acts of conduct by the licensee similar to acts or conduct that would constitute grounds for action as defined in this section, a certified copy of the record of the action taken by the other state or jurisdiction being conclusive evidence thereof.

(32) Failure to report to the Board any adverse action taken against him or her by another licensing jurisdiction (United States or foreign), by any governmental agency, by any law enforcement agency, or by any court for acts or conduct similar to acts or conduct that would constitute grounds for action as defined in this section.

(33) Failure to report to the Board surrender of a license or other authorization to practice medicine and surgery in another state or jurisdiction, or surrender of membership on any medical staff or in any medical or professional association or society while under disciplinary investigation by any of those authorities or bodies for acts or conduct similar or to acts or conduct that would constitute grounds for action as defined in this section.

(34) Any adverse judgment, award, or settlement, or award arising from a medical liability claim related to acts or conduct similar to acts or conduct that would constitute grounds for action as defined in this section.

(35) Failure to transfer pertinent and necessary medical records to another physician in a timely fashion when legally requested to do so by the subject patient or by a legally designated representative of the subject patient.

(36) Improper management of medical records.

(37) Failure to furnish to the Board, its investigators or representatives, information lawfully requested by the Board.

(38) Failure to cooperate with a lawful investigation conducted by the Board.

(39) Violation of any provision(s) of the medical practice act or the rules and regulations of the Board or of an action, stipulation, or agreement of the Board.

(40) The inability to practice medicine and surgery with reasonable skill and safety to patients by reason of age; illness; drunkenness, excessive use of drugs, narcotics, chemicals, or any other type of material or as a result of any mental or physical condition. To enforce this paragraph, the Board may, upon probable cause, request a physician to submit to a mental or physical examination by physicians designated by it. If the physician refuses to submit to the examination, the Board shall issue an order requiring the physician to show cause why he will not

"You are not qualified to determine the future of medicine"

The Politician Versus the Professor

"The history of the world is but the biography of great men," according to Thomas Carlyle, and a historical fillip surfaced recently as correspondence between two influential men became public.

President Harry Truman, who had the political courage to start the Atomic Age, exchanged letters with Oklahoma physician Lewis J. Moorman on the government's role in the delivery of medical care. These letters are in the Robert M. Bird Library archives at the University of Oklahoma Health Sciences Center and offer a tantalizing peek at the thought processes of two very influential men who were at their apex of power a half-century ago. It is intriguing that they wrote on a subject that still roils the nation, still divides policymakers, and still causes tension and unrest among the citizens.

A half-century ago, Lewis J. Moorman, MD, was a towering and respected figure in Oklahoma medicine. With a long and most distinguished medical practice career in both rural and metropolitan Oklahoma, he was a true medical leader, educator, and thinker. Dr. Moorman served as dean of the medical school, president of the Oklahoma State Medical Association, the National Tuberculosis Association, the American Trudeau Society, the Southern Medical Association, and as editor-in-chief of the OSMA JOURNAL. He was an internationally recognized expert on tuberculosis. His book *Pioneer Doctor* brought national acclaim for his insight and heart.

In 1947, both Great Britain and the United States were debating the role of governments in the delivery of health care. With a strong Socialist movement present, Great Britain proceeded with their National Health Service, activating it the next year. Simultaneously, in the U.S., the Truman Administration proposed the

"Public Health Program" and Dr. Moorman wrote to President Truman as follows:

July 7, 1947

Dear Mr. President:

According to the press, in your messages and public utterances proclaiming the need of compulsory health insurance, you have employed the words "compulsion" and "freedom" as though they possessed a certain degree of kinship and as if they might work together in friendship. According to my interpretation of Mr. Webster, the two words are mortal enemies and can never be successfully harnessed in the same cause. Throughout the ages slaves would have been free except for compulsion.

Recently the world was shocked by the revelation of mass medical murder in Germany under the Hitler regime. This could never have happened if Bismarck had not clandestinely murdered the free spirit of medicine and of the people through his social security program including compulsory health insurance. The lathe of so-called social security turns out a world of trouble and unrest. One hundred years of compulsion in Germany destroyed professional freedom, robbed the individual doctor of personal liberty, and left him without a choice between right and wrong. Because of the inferior quality of regimented medicine, it represents insidious mass murder which get under way as soon as any compulsory health insurance in instituted, and becomes vulnerable to open mass murder when democracy is destroyed by so-called social security and Government paternalism.

The people of your great Middle West remained at home with their ears to the ground when you went to Congress. Surrounded by the last vestiges of unadulterated Americanism, they have heard the confused rumbling of democracy on the road to serfdom, and as you entered the White House they wondered if you would remain true to the spirit of your section so representative of American freedom. Here, the people are vigorous, competent, generous, and happy because of free enterprise.

Your advocacy of regimented medicine has surprised and disappointed the best of your compatriots. In this Bismarckian business there is abysmal darkness from which politicians may never return. You may know your politics but you are not qualified to determine the future of medicine. You do not know how to follow the path that nature walks, nor do you know how to direct the art of healing. The social security director, though naively willing, is not qualified to serve as family physician to one hundred and forty million people.

> In this Bismarckian business there is abysmal darkness from which politicians may never return."
>
> —*Lewis J. Moorman, MD*

May I call your attention to the fact that we used to go abroad to learn medicine, and that at time passed regimentation of European doctors weakened the appeal of the world's great clinics and medical schools and finally they were virtually destroyed by the War. Now the world is looking to America for the advancement of medical learning and postgraduate education. Regimentation of the medical profession and the people of this country through compulsory health ("sickness") insurance would rob the United States of a great opportunity to serve a sick world.

Trusting that you may give serious attention to the history of medicine in this country and the results of compulsory health insurance in other comparable nations before establishing controls and limitations which would seriously alter the course and quality of medical service in the United States, I am,

Very sincerely,
Lewis J. Moorman, MD

President Truman's terse reply suggests that he knew little of Dr. Moorman's studiousness, or perhaps that he may have been piqued at the challenge to his qualifications to determine the future of medicine:

July 16, 1947

Dear Dr. Moorman:

I read your letter of July seventh with some surprise.

(continued)

submit to the examination and shall schedule a hearing on the order within thirty (30) days after notice is served on the physician. The physician shall be notified by either personal service or by certified mail with return receipt requested. At the hearing, the physician and his attorney are entitled to present any testimony and other evidence to show why the physician should not be required to submit to the examination. After a complete hearing, the Board shall issue an order either requiring the physician to submit to the examination or withdrawing the request for examination. The medical license of a physician ordered to submit for examination may be suspended until the results of such examination are received and reviewed by the Board.

(41) Failure to provide a proper setting for medical act, including but not limited to examination, surgery, or other treatment.

(42) Failure to inform the Board of a state of physical or mental health of the licensee or of any other health professional which constitutes or which the licensee suspects constitutes a threat to the public.

(43) Failure to report to the Board unprofessional conduct committed by another physician.

[Source: Amended at 9 Ok Reg 1579, eff 4-27-92; Amended at 10 Ok Reg 1529, eff 4-26-93; Amended at 10 Ok Reg 4375, eff 7-27-93 (emergency); Amended at 11 Ok Reg, eff 4-4-94 (emergency)]

Use of mouthguard prevents oral trauma

Every athlete who participates in a contact sport is at risk of sustaining an orofacial injury. Mouthguards, or mouth protectors, provide protection against injuries to the orofacial area, including the teeth, lips, cheek, and tongue, thereby reducing the incidence and severity of injuries that occur during athletic practice and competition. Mouthguards also have been shown to reduce the incidence of head and neck injuries, concussions, and jaw fractures.

There are three basic types of mouthguards: (1) stock mouthguards, such as those purchased in sporting good stores, which are acceptable but are often loose and cumbersome because they are not formed to the mouth and teeth: (2) mouth-formed mouthguards, also known as "boil and bite" mouthguards, which have thermoplastic linings that adapt to the teeth and are the most popular type of mouthguard (used by more than 90% of the athletic population); and (3) custom fabricated mouthguards, which are made by a dentist and are the best mouth protectors from the standpoint of fit, retention, comfort, and ease of speech, and also have the highest level of player acceptance (a quarterback on a football team, for example, would have to have a custom fabricated mouthguard so that he can call signals and direct the team). Mouthguards are generally worn only on the maxillary teeth.

Organizations with recreation and sports programs, as well as those that provide space, equipment, or facilities for sports, can play a significant role in reducing injuries and traumas by requiring the use of appropriate protective gear. In 1962, the use of mouthguards become mandatory for high school football. In 1974, the NCAA made mouth protectors and face guards mandatory for football,

The Politician *(continued)*

It is perfectly apparent that you are not familiar with the Public Health Program advocated by this Administration.

I am sorry that you haven't taken the trouble to enlighten yourself on the subject.

Sincerely yours,
Harry Truman

Dr. Moorman's second letter obliquely reminds President Truman of the Administration's considerable turmoil on the labor front, but again encourages a free enterprise solution in health delivery:

September 9, 1947

My Dear Mr. President:

It was good of you to take time to answer my letter but a bit unfair to summarily dismiss my patriotic fears for the people of our great country with the assumption that I am not informed about the Administration's proposed Public Health Program.

Fortunately, I have enjoyed living in a free country and serving my people through free enterprise. With no uninformed bosses in Washington and no directives, bulletins, and blanks to curtail my time which has been wholly devoted to patient welfare and professional attainments. This life work represents my small contribution to my country. Recent trends toward socialism which I consider government paternalism, have caused me to add the threat of compulsory health insurance to my studies in order that it may have fair consideration in the light of what I know of patients and medical practice.

Please note that I have not added all national and international problems as you have had to do. I am sorry for you. Even a rare genius could not encompass all with adequate comprehension.

Why don't you look over the fine record of American medicine, compare it with any other countries and be comforted by its relative rating and its present high health level and falling mortality rates? In the face of your troubles, including the threat of another war, why not leave medicine, one of the few going concerns in the United States, alone? Physicians sponsor no strikes, pose no picket lines and seldom turn the poor away. They come to Washington only in behalf "of the people, by the people and for the people." If war should come, again they will volunteer and again they will give our combatants the best care the medical world has to offer.

Being president of the United States entails a frightful responsibility. In response to your consciousness of the responsibility, undoubtedly you must be thinking not only of the present but of the future.

Humbly, I claim enough knowledge of medicine and medical needs to know that you will save face while you are living and add stature to your future if you consider medicine a free enterprise and leave it in the capable hands which have brought us to our present high health level.

Trusting you will pardon this earnest expression of the faith within me I am,

Sincerely,
Lewis J. Moorman, MD

hockey, and lacrosse. Few organized leagues and intramural sports require protective gear. The exceptions are football, boxing (which requires mouthguards), and collegiate and under-18 ice hockey, which require players to wear face guards, helmets, and mouthguards.

It has been demonstrated that requiring the use of protective headgear can considerably reduce injuries among those involved in professional and amateur sports, including basketball, football, ice hockey, field hockey, baseball, softball, lacrosse, soccer, squash, racquetball, wrestling, boxing, and gymnastics. For example, the use of mouth protectors and face guards in organized football reduced the incidence of mouth and face injuries from 50% of all football injuries during the 1950s to about 0.4% today. In contrast, 10% of baseball and 13% of ice hockey injuries are to the mouth and face.

Unfortunately, mouth protectors and face guards are not currently required in most sports. Consequently, health professionals should work together to make sure that regulations are passed that require the use of appropriate protective gear.

HIV/HBV home infection control pamphlet is now available

The HIV/STD Service of the OSDH has recently published a new pamphlet titled *Infection Control in the Home: Protecting Yourself from HIV and HBV.* The pamphlet provides information on measures which should be taken to prevent transmission of HIV or hepatitis B to family members and friends providing personal care to infected persons. The department urges all health care providers to make the pamphlet available to infected patients and their caregivers, as appropriate. The pamphlet is available through the Films and Publications Division of the Oklahoma State Department of Health by calling (405) 271-5724.

LETTERS

AHCPR guides oversimplify treatment approach

To the Editor: The May 1994 JOURNAL has an excellent review article by Dr. Marie Bernard in which stress and urge incontinence treatment protocols are presented and discussed. The AHCPR guideline algorithms that are presented seem straight forward and easy to follow; however, in practice the situation is not so clear. Those of us in urology and gynecology who have literally "gotten our feet wet" in the war against incontinence have developed a philosophy of treatment to go along with such algorithms. One major philosophic principle is to set treatment outcome goals to be met. Progress toward these goals needs to be followed since behavioral therapy does not change anatomical problems and thus does not work well with stress incontinence. Urge incontinence improves but usually does not resolve completely, leading to the adjunct use of drugs. This in turn can cause polypharmacy problems, side effects, and ultimately noncompliance. But remember, a wet woman that is better but is not cured is still a wet woman. So if treatments are not curative, further therapy is definitely indicated.

Sorting out the incontinence etiology in elderly females can be more difficult than one might suspect, since most will have mixed incontinence and many will also have nonobstructive increased residual urine

(continued)

There is no evidence that this exchange of letters affected the thinking of either of these leaders, but the Truman Administration's health proposals ran into difficulty in Congress. Several more incremental health initiatives were promoted by the Washington Socialists until Medicare was enacted in 1965. The then ex-President Truman traveled to the Capitol to be presented the first Medicare cards with considerable fanfare that recognized his pivotal role in socializing the medical care of the elderly.

It is remarkable that a half-century later the struggle between Socialism and free enterprise continues despite the interim worldwide discrediting of socialistic platforms. The ancient conflict between citizen freedom and government compulsion continues even now as the present Administration tries to establish compulsory "medical alliances" on yet another group of U.S. citizens. The verbal joust between Dr. Moorman and President Truman may illustrate the point that while the pen may be mightier than the sword, ideas can require many generations to prevail against the political power brokers of statism.

—*Ray V. McIntyre, MD*

We thank the archives of the Robert M. Bird Library, University of Oklahoma Health Sciences Center, Oklahoma City, for the copies of the letters and Dr. Wilson "Dave" Steen for their retrieval.

LETTERS (CONTINUED)

as well. While surgery is usually required to correct the anatomical problems in stress incontinence, it usually does not correct urge incontinence and can make residual volumes worse. Anticholinergics for urge incontinence can likewise make residual urines worse. This trade off of simultaneous good and bad results with each treatment often leads us to use long-term catheterization. Clean intermittent catheterization has the lowest complication rate but is the hardest to effectively continue long-term. While urethral foley placement is easy to do, it has the most complications. Suprapubic tube placement is usually the best compromise long- term when other catheterization treatments are difficult or fail. Complete urinary diversion should seldom be required.

Setting a treatment goal of complete dryness in these elderly females often requires considerable effort of the primary care physician as well as consulting specialists, but the outcome may well be worth it for these wet and often miserable women.

—G. Douglas Corrie, MD
Urologist, Oklahoma City

DEATHS

Max A. Glaze, MD
1923 - 1994

Max A. Glaze, MD, retired Muskogee psychiatrist, died April 29, 1994. A native of Tonkawa, Dr. Glaze was a graduate of Ponca City High School, Tonkawa Junior College, and the University of Oklahoma School of Medicine, where he received his medical degree in 1951. An OSMA Life Member and a fellow in the American Psychiatric Association, Dr. Glaze was in active practice from 1959 to 1987

Edward David Greenberger, MD
1907 - 1993

Longtime McAlester radiologist Edward D. Greenberger, MD, died August 6, 1993. The OSMA awarded him Life Membership status in 1980, and a fifty-year pin in 1982 to commemorate his half century in practice. A native of Passaic, N.J., Dr. Greenberger earned his medical degree at New York University Medical College in 1932. Two years later he established one of the state's first radiology practices in McAlester. During World War II, he interrupted his practice to serve with the U.S. Army for three years, including 18 months in Europe. Dr. Greenberger was a member and past president of the Oklahoma Radiological Association, a member of the Masonic Lodge, a 50-year member of the Rotary, and a fellow of the American College of Radiology.

Carter William Mathews, MD
1935 - 1994

Obstetrician-gynecologist Carter W. Mathews, MD, died June 3, 1994, in Ponca City, the town where he was born. Dr. Mathews earned his medical degree from Tulane University School of Medicine in 1962 and completed his residency at Oakland Naval Hospital in California. After completing another two years of military service at the naval hospital in Quantico, Virginia, he returned to Ponca City to practice.

Winfred Aaron Showman, MD
1896 - 1994

Dr. Winfred A. Showman, a practicing dermatologist in Tulsa for more than 50 years, died May 14, 1994. A veteran of World War I, he was graduated from Washington University School of Medicine, St. Louis, Mo., in 1921. Dr. Showman was a fellow of the American Academy of Dermatology. In the course of his career he served as president of the Tulsa County Medical Society, section chairman for dermatology for the Southern Medical Association, and foreign ambassador for the American Medical Society. In 1971 a dermatology professorship was established in his name at Washington University. Dr. Showman co-authored more than 100 publications on dermatology research, published five dermatology textbooks, and presented almost thirty abstracts.

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1993

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Edward Cotter Murray, MD ..... April 9
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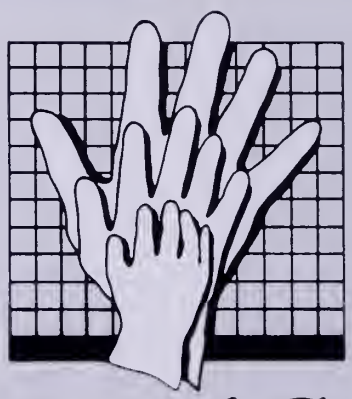

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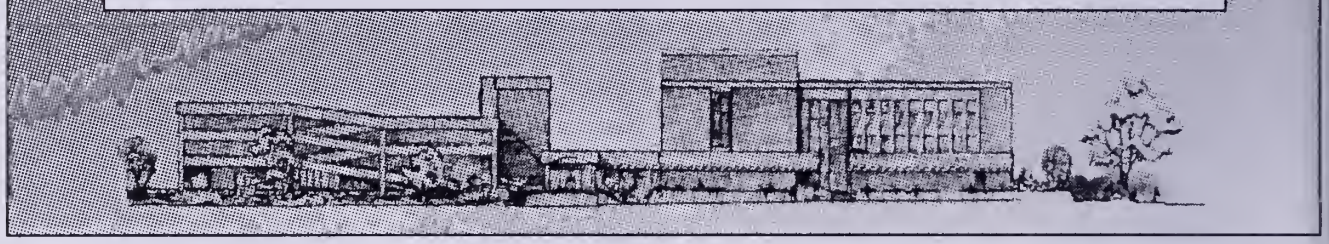

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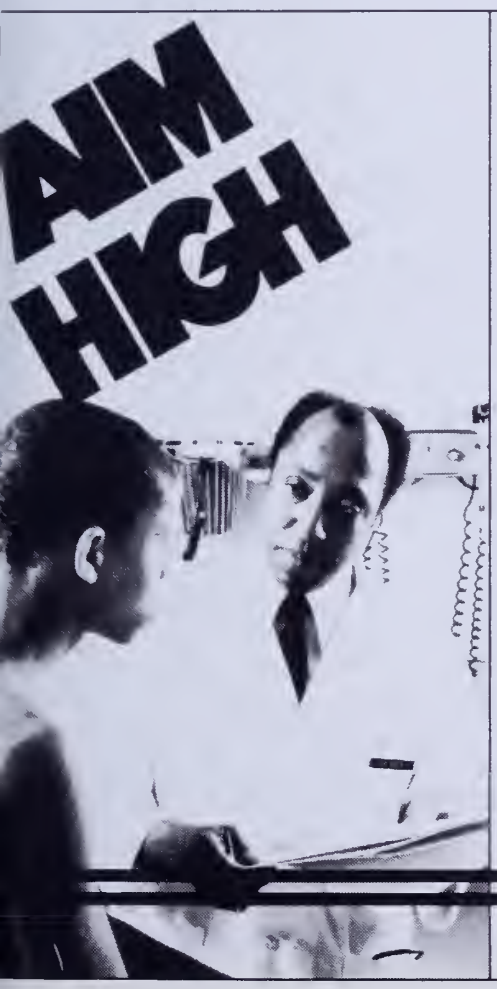

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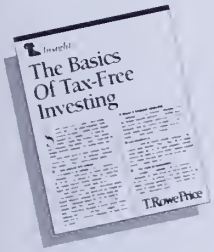

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The Last Word

■ **Oklahoma City pathologist Perry A. Lambird,** MD, announced in Chicago last month his candidacy for the office of vice speaker of the American Medical Association House of Delegates. The election itself will take place at the AMA's annual meeting in Chicago next June. Dr. Lambird is a past president of both the Oklahoma State Medical Association and the Oklahoma County Medical Society. His candidacy for a national AMA office is the first by an Oklahoman since 1967, when Malcolm E. Phelps, MD, El Reno, was elected AMA vice president.

■ **The OSMA Council on Medical Service will** be working closely with the Oklahoma Legislature in studying the administration of the state employees' insurance plan by the Oklahoma State Employees Group Insurance Board (OSEEGIB). As part of the information gathering process, OSMA members are being asked to report to the Council on Medical Service their ten most frequently used CPT codes, their normal charges, and both their 1993 and 1994 OSEEGIB reimbursement levels. Information should be sent to the OSMA Council on Medical Service, 601 Northwest Expressway, Oklahoma City, OK 73118. Reporting forms were provided in the June issue of *OSMA News*.

■ **Robert W. Baker III, Tulsa, OSMA associate** director for 10 years and director of the Oklahoma Medical Political Action Committee (OMPAC), has resigned his position to pursue other interests. Baker will serve as finance director for the Cody Graves campaign. Graves is running for the office of U.S. Senator in the Democratic primary in August and will face Rep. Dave McCurdy.

■ **Only 39% of Oklahoma County two-year-olds** receive all their immunizations, says Dr. Jo Ann Wine, pediatrician and director of the Children's Immunization Station at Children's Hospital of Oklahoma. Dr. Wine says parents tend to wait until their children are about to enter school before having them immunized. Consequently, the threat of polio and diphtheria is increasing and there have been recent outbreaks of measles and whooping cough. Failure to vaccinate early is to blame. Children should receive their first DPT (diphtheria, pertussis, tetanus) shots at 2 months of age, as well as HIB (*Hemophilus influenza b*) and OPV (oral polio vaccine). At 12 to 15 months, they should receive their first rubeola (measles), mumps, and rubella (MMR) vaccinations. "To facilitate the schedule and to help bring the immunization rate closer to 90% by 1996, the Immunization Station offers free shots and booster shots to any child," says Dr. Wine. "Children's records are put on computer, and parents are reminded when they need to return." Walk-in appointments are welcome. The station, open from 9 am to 5 pm each weekday, is located at Children's Hospital, 940 N.E. 13th Street, Oklahoma City, on the first floor of Bielstein Center.

■ **Lynn V. Mitchell, MD, assistant professor of** occupational and environmental medicine at the University of Oklahoma Health Sciences Center (OUHSC) and medical director of the Student Health Service, is among 25 physicians named to the 1994 Class of Fellows and Masters in the American College of Occupational and Environmental Medicine (ACOEM). The international organization has some 7,000 physician members who promote worker and environmental health through preventive medicine, clinical practice, research, and education. Dr. Mitchell was named a Master, indicating a demonstrated commitment and proficiency in occupational and environmental medicine and to the future of the college.

■ **The Oklahoma Board of Medical Licensure** and Supervision has set the following meeting dates for the remainder of 1994: July 22, hearings; July 23, business; September 23-24, hearings; November 18, hearings; November 19, business.

■ **OU College of Medicine Professor Edward N.** Brandt, Jr., MD, has received the 1993 American College of Healthcare Executives Student Chapter Outstanding Faculty Member Award for teaching excellence. Dr. Brandt, holder of both MD and PhD degrees from the University of Oklahoma, is co-director of the OUHSC Center for Health Policy. He joined OU as executive dean of the College of Medicine in 1992 after serving as president and professor of medicine of the University of Maryland at Baltimore from 1985 to 1989. ❑

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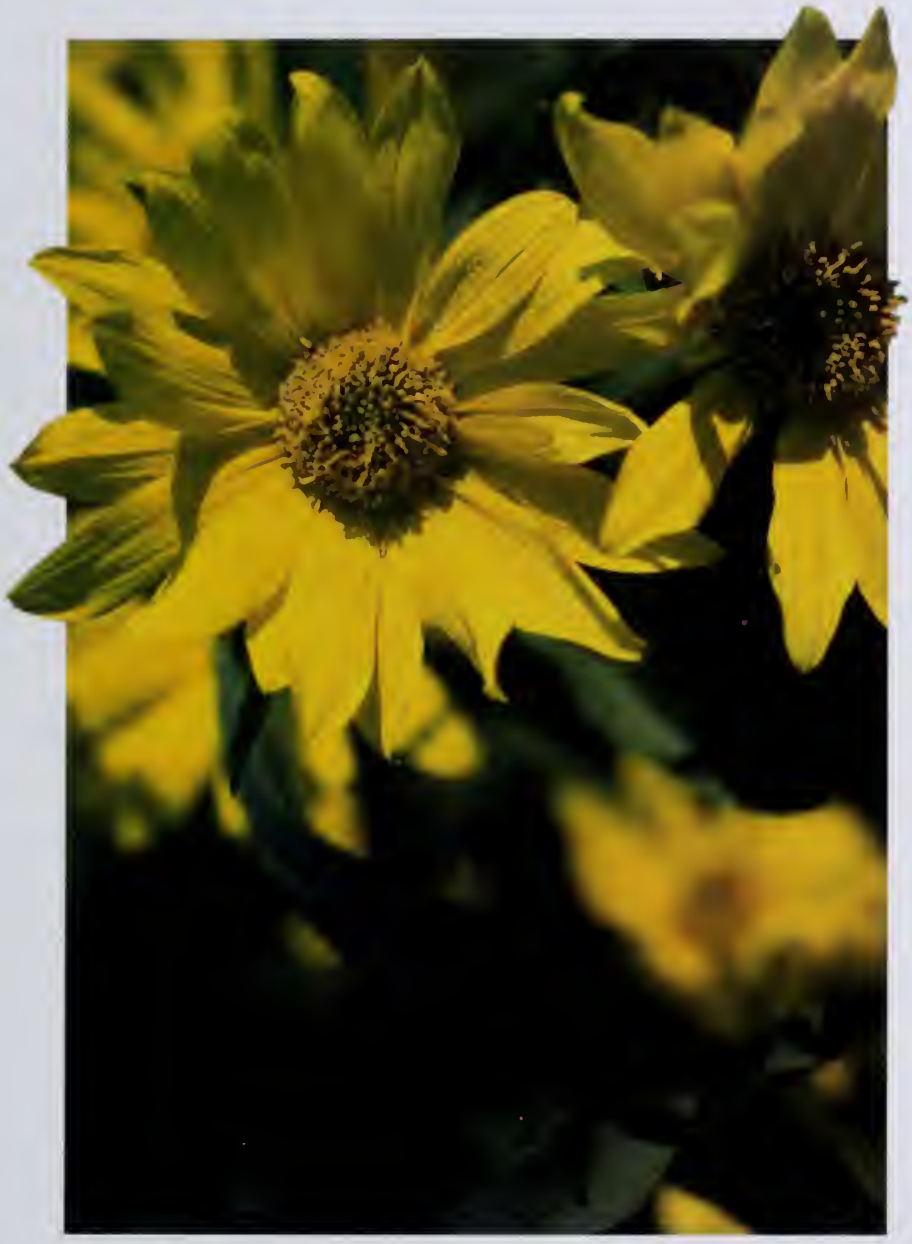

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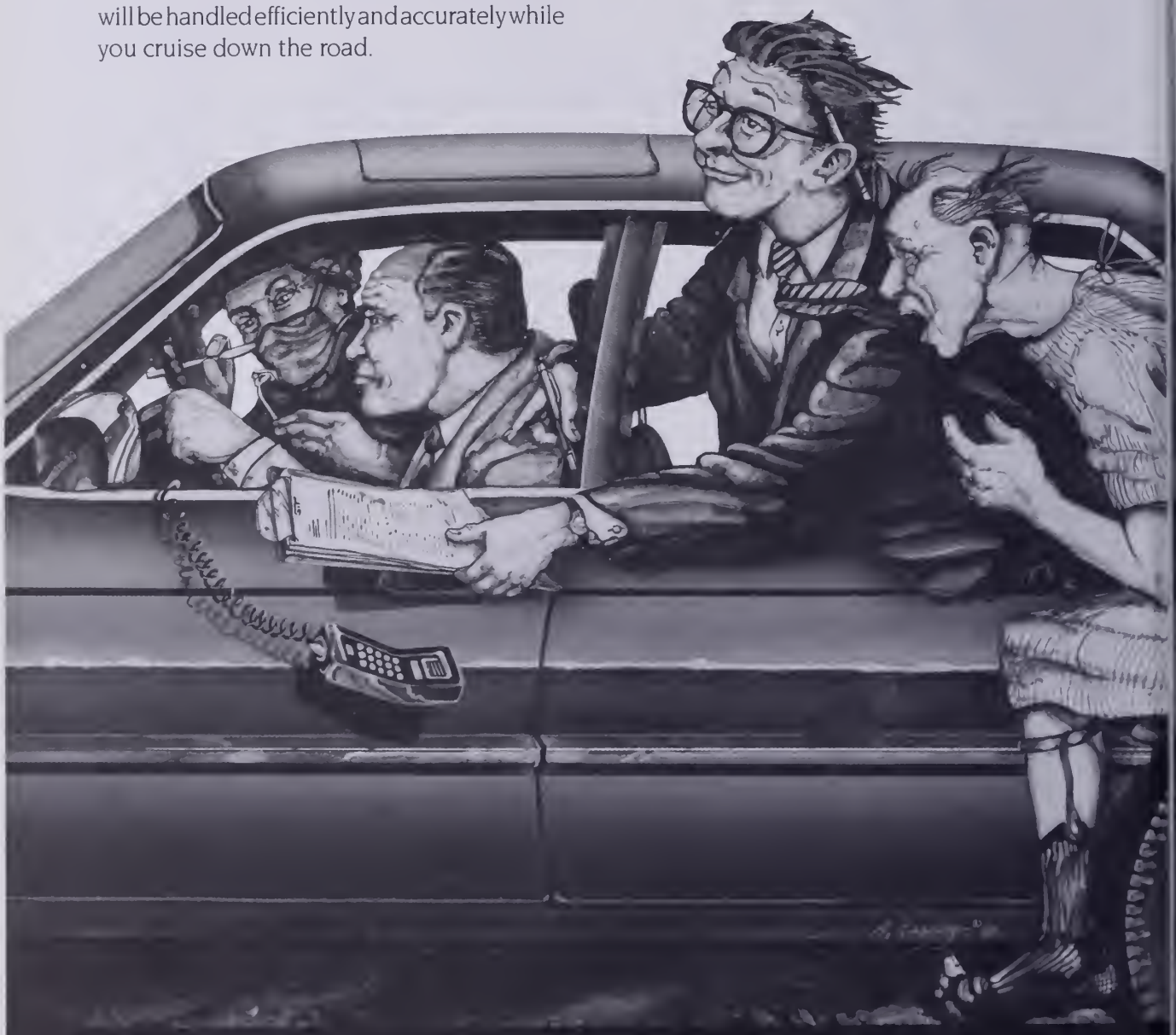

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The JOURNAL (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125. POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single issues are $3 per copy, prepaid, subject to availability.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

AUGUST 1994 VOL. 87, NO. 8

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GUEST EDITORIAL

Clifford G. Wlodaver, MD

Why Have Autopsies Grown So Unpopular and What Can We Do About It? The Clinical-Pathological Conference!

Knowing a patient's diagnosis is the pillar of clinical medicine. Without it, management of a patient's illness must be empiric, perhaps misguided. True, with the never ending technological advances, we are becoming better diagnosticians. Yet some patients defy even our best diagnostic efforts and succumb, without a diagnosis and without potentially life-saving treatment.

Enter, the postmortem examination. While the deceased patient benefits not, we clinicians may be the smarter when confronted with the next similar case. *Hic locus est ubi mors gaudet succurrere vitae* ("This is the place where death delights to help the living"). So why have autopsies grown so unpopular among physicians?

There is a common misperception that nothing more can be learned, and that even if an unsuspected disease were uncovered, the discovery would be of no benefit. On the contrary, the incidence of important surprise findings can be significant[1] and can be used for quality control.[2] Then there is the fear of uncovering a missed diagnosis that leads to malpractice litigation. However, there is no evidence that physicians who ask for autopsies are sued more often than those who don't.[3]

And regrettably, many pathologists themselves are opposed to autopsies, not because of clinical disinterest but because of financial disincentives and, alas, fear of AIDS. Autopsies, while quite time-consuming, are not reimbursed. (Perhaps this inequity can be corrected along with other healthcare reforms.)

As for the fear of AIDS, it ranks high among pathologists' reluctance to perform autopsies.[4] In fact, known AIDS cases are outright refused autopsies by some pathologists (personal experience). While the risk of contracting HIV from a percutaneous injury is approximately 0.4%[5] and no pathologist has been infected in this manner, the irrational demand for "zero risk" is invoked. When clinicians and families desire an autopsy on a patient who has died of AIDS but are refused by the pathologist, we have a particularly lamentable situation. AIDS and its complexities are among the diseases we know least about, especially in Oklahoma, where AIDS is relatively uncommon. Patients may die prematurely of opportunistic infections that were undiagnosed and possibly even unsuspected. Although a postmortem examination on one patient will not change the ultimate outcome of HIV disease in any other patient, it may add quality time to a life or two, or at least reduce the suffering if it leads to more rapid diagnosis.

We all have lost patients, probably friends and family as well, at relatively young ages. As we grieve and listen solemnly at funerals, the reverberating question is *why?* Beyond the religious and metaphysical answers, we as physicians are empowered to respond. And possibly one patient's untimely death—if better understood from the postmortem exam—can breathe life into another.

What can we do to resuscitate the autopsy? It goes hand in hand with the clinicopathological conference. Promoting interest in and returning this exercise to the agenda at all hospitals, whatever the size and staff, can only lead to improving our diagnostic skills for the living.

—C.G. Wlodaver, MD

Acknowledgment

I am grateful to Dr. John Harkess for reviewing the manuscript.

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Jay A. Gregory, MD

Have a Good Day...

There is so much to say, and so little time and space in which to write. So read carefully, my friends; our future will depend on our actions.

What would it take for you to have a good day, professionally speaking? If you could answer that question, then you could focus your efforts on what it would take to accomplish a good day. Makes sense, doesn't it? The trite phrase "have a good day" was obviously originated in sunny Southern California by the beach bums of the 60's and was meant as sort of a slam, in that if you didn't have a good day, it was your fault. And basically the same holds true today. Too many times we are simply adrift upon the seas of life and probably deserve whatever happens to us. The obvious corrective action is to *carpe diem*. Literally, we must seize the day.

On a good day, all patient expectations would be met or exceeded. The OR schedule would go smoothly, without a hitch. Patient flow through the office would be perfectly smooth. There would be time at the end of the day to do the paperwork. And, you would be home in time for supper with the family. (At least it is something to shoot for.) To fulfill such a dream, each reader of this article must individually and collectively do her or his part to accomplish such a dream. Utopia exists only in your mind. You create your own world. And only you can make those changes in your daily activities which determine whether you live in heaven or hell.

If most of you are like me, and I will assume that you are, then your office is the only little spot where you can escape, momentarily. Of course, that assumes you have in place mechanisms which allow you to complete all of those tasks associated with a busy office practice; otherwise, you will always be desirous of escaping from the office, an entity which you created.

Which brings me to the entity we are about to create. Let me describe for you some of the characteristics our IPA-MCO should have to help us have a good day every day.

First of all, we must remember that the IPA-MCO is there for us, whereas elsewhere, doctors are there for the plan. You may criticize or disagree with this statement and say that patients come first, and I agree. But for our immediate and long-term survival, the IPA-MCO exists for us, to serve us and our patients' needs.

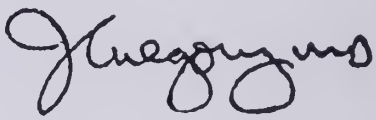
Secondly, if we are to be successful, at least five crucial requirements must be met: (1) We cannot afford to be undercapitalized. (2) We must have the participation of as many physicians across the state as possible. (3) Quality, quality, quality must be maintained in the product we deliver. (4) Good administrators must be given good direction by a good board of directors who have been elected by the democratic process. (5) Everybody must be a team player.

Plans that have failed in the past have been undercapitalized, have lacked a business plan, and have not been operated with the proper business attitude. Your chosen leaders, with the assistance of topnotch administrators, will develop a good business plan. Those of you chosen to be directors must develop a good business attitude and conduct yourselves and the business of the IPA-MCO in such a manner. And only you, by your participation, can ensure that the plan will not be undercapitalized.

Totally doctor-owned plans can protect the physicians and patients from the bottom-line mentality that characterizes the commercial ventures in existence today. Even as you read these words, alliances are forming in this state with the sole purpose of controlling you and your patients. Many of you have already been engulfed by an alliance in an effort to get what you can while the getting is good. You are possibly helping yourself in the short run, but at the expense of your patients and, most importantly, your colleagues and your profession.

The cash out-of-pocket will not be big bucks per individual. We are not doing this to make a big return on investment. We are doing this in order to be able to continue making a living doing what doctors have always done.

Have a good day!!!



SCIENTIFIC

Understanding Mechanical Ventilation: Indications for and Initiation of Therapy

Brent R. Brown, MD

The growing complexity of mechanical ventilators and the proliferation of new ventilator modes may confuse and intimidate clinicians who on occasion must manage a mechanical ventilator. The fundamental indications for and types of mechanical ventilation are reviewed, and guidelines for basic ventilator settings are provided. By understanding and applying these essentials, the clinician can provide effective mechanical ventilation to most patients who require it. A subsequent article published in this journal will review the related topics of patient monitoring during mechanical ventilation, complications of mechanical ventilation, and weaning from mechanical ventilation.

Direct correspondence to Brent R. Brown, MD, University of Oklahoma Health Sciences Center, Department of Medicine, Post Office Box 26901, Room 3SP 400, Oklahoma City, OK 73190.

THE PHYSICIANS of one Oklahoma tertiary care hospital were recently surveyed concerning their continuing medical education needs. Surprisingly, mechanical ventilation was the single most frequently requested topic for further education. Whether the results of this survey apply to physicians in other practice settings in our region is a matter of conjecture. However, many primary care physicians, surgeons, and specialists in urban as well as rural areas are occasionally thrust into situations where they must manage a mechanical ventilator. The fact that they infrequently operate mechanical ventilators may create considerable anxiety. The increasing complexity of the latest generation of ventilators and the plethora of new ventilator modes described in medical journals can be intimidating.

The good news for practitioners is that the basic principles of mechanical ventilation are relatively straightforward and have changed little over the past decade. This article provides practical information on the indications for mechanical ventilation, discusses basic ventilatory modes, and provides guidelines for the application of positive pressure ventilation for adults. A subsequent article will focus on the issues of patient monitoring during mechanical ventilation, complications of mechanical ventilation, and weaning from mechanical ventilation.

Indications for Mechanical Ventilation

The major indication for mechanical ventilation is acute respiratory failure. Mechanical ventilation is also used during general anesthesia and in selected cases of chronic, irreversible respiratory failure. Acute respiratory failure is characterized by one or both of the following problems: impaired oxygenation or hypoxemia, and inadequate ventilation or hypercapnia (Table 1). Impaired oxygenation most commonly results from mismatching of ventilation ($\dot{V}$) with perfusion ($\dot{Q}$) in diseased areas of the pulmonary parenchyma. Right-to-left shunting of blood due to intracardiac or intrapulmonary pathology and conditions causing hypoventilation may also result in hypoxemia. $\dot{V}/\dot{Q}$ mismatching, shunting, and hypoventilation occur concurrently in some situations. Respiratory failure due to pneumonia, pulmonary emboli, the adult respiratory distress syndrome (ARDS) and hydrostatic pulmonary edema is primarily a problem of hypoxemia, although hypercapnia may ensue in patients with severe disease.

Ventilatory failure, a second form of *acute* respiratory failure, is indicated by an elevation in arterial CO_2 tension (P_aCO_2) and a low arterial pH. Ventilatory failure can also be a form of *chronic* respiratory failure where hypercapnia is associated with an elevated serum

Table 1. Classification of Acute Respiratory Failure

Types	Pathophysiology	Clinical Examples
Failure of Oxygenation (Hypoxemia)	Ventilation/ Perfusion Mismatching	—Exacerbation of asthma —Pneumonia
	Right to Left Shunt	—Adult respiratory distress syndrome —Hydrostatic pulmonary edema
Failure of Ventilation (Hypercapnea)	Hypoventilation —Reduced respiratory drive (won't breathe)	—Drug intoxication —Head trauma, cerebral vascular accident
	—Impaired respiratory pump function (can't breathe)	—Respiratory muscle fatigue —Neuromuscular disease
	Increased Dead Space	—Emphysema —Pulmonary embolus
	Increased CO_2 Production	—Increased work of breathing (any cause) —Fever —Excessive carbohydrate intake

Table 2. Indications of Potential Need for Mechanical Ventilation

Deteriorating Bedside Findings	—Obtundation —Progressive tachypnea —Progressive use of accessory muscles of respiration
Progressive Hypercapnea	—Rising P_aCO_2 with pH < 7.30
Progressive Hypoxemia	— P_aO_2 < 65 mm Hg on an O_2 concentration > 50%

Table 3. Classification of Mechanical Ventilators

Type	Example
Negative Pressure	Iron Lung, Curirass
Positive Pressure	
—Pressure Cycled	Bird Mark 7, 14
—Volume Cycled	Puritan-Bennet and Bear models
—Time Cycled	Seimens Servo 900, 300

bicarbonate and a normal pH. *Acute* ventilatory failure can be superimposed on *chronic* ventilatory failure, as is sometimes seen in exacerbations of chronic obstructive pulmonary disease (COPD). Hypoventilation occurs in patients when they "won't breath" because of a reduced respiratory drive or when they "can't breath" due to pathology of the lungs, chest wall, or neuromusculature. Acute reductions in respiratory drive may be due to drug intoxications, head trauma, cerebrovascular accidents, or other intracranial catastrophes. Hypoventilation may occur in patients with an intact respiratory drive who "can't breath" due to severe kyphoscoliosis, muscular dystrophies, Guillain-Barré syndrome, or other diseases of the chest wall or neuromusculature. The most common subset of patients in the "can't breath" group are those who develop respiratory muscle *fatigue* secondary to the increased work of breathing associated with severe lung disease of any type. Combinations of hypercapnic and hypoxemic respiratory failure are common in this last subset of patients. Increases in lung dead space volume or increases in CO_2 production are additional pathophysiologic pathways which can contribute to ventilatory failure in some conditions (Table 1).

No universally accepted criteria for the initiation of mechanical ventilation exist, making the decision to institute therapy a clinical judgment that must be individualized for each patient. Some potential indications for mechanical ventilation are listed in Table 2. Serial examinations assessing the severity of respiratory distress and periodic arterial blood gas measurements are useful in following the course of a patient that may require mechanical ventilation. Progressive tachypnea, increasing use of accessory muscles of respiration, or worsening supraclavicular or intercostal retractions despite appropriate treatment warrants intubation and mechanical ventilation in most situations. Many experts recommend initiation of mechanical ventilation for rising $PaCO_2$ values in association with a pH < 7.30, particularly if rapidly reversible causes of ventilatory failure are not obvious.[1] In hypoxemic respiratory failure, a persistent P_aO_2 less than 65 mm Hg despite high flow oxygen at concentrations exceeding 50% has also been cited as justification for mechanical ventilation.[2] Ideally, the patient should understand the risks and benefits of mechanical ventilation and agree to proceed. Occasionally, mechanical ventilation must be instituted for unresponsive patients without established limits of care under the assumption that the patient would desire this therapy if able to provide consent.

Types of Mechanical Ventilators

Ventilators are classified as either posi-

tive pressure or negative pressure designs based on the process they use to inflate the lungs (Table 3). Negative pressure ventilators include the iron lung and the cuirass and are not routinely used for the treatment of acute respiratory failure. Positive pressure ventilation has become the treatment of choice for both acute and chronic respiratory failure. Positive pressure devices can be subdivided into pressure cycled, volume cycled, or time cycled designs depending on how they terminate the inspiratory phase of the respiratory cycle. Pressure cycled devices are currently used to deliver intermittent positive pressure breathing (IPPB) treatments, but are not recommended for use as mechanical ventilators. Volume and time cycled ventilators have several advantages over earlier pressure cycled designs and numerous models of both types enjoy widespread use.

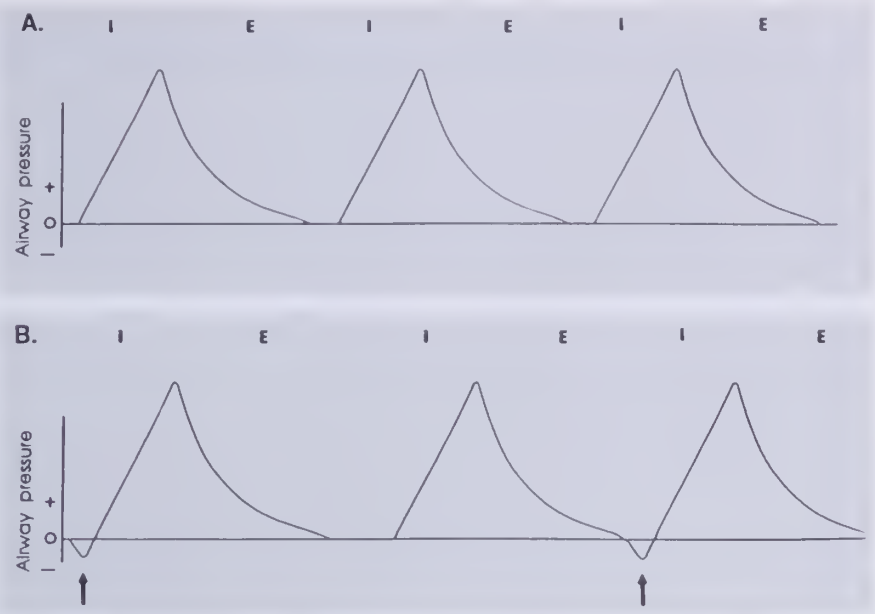


Figure 1. Airway pressure versus time wave forms for control mode (A) and assist-control mode (B). I indicates inspiration and E expiration. The patient is unable to alter the respiratory pattern with control mode. The patient may trigger extra breaths with assist control, but controlled breaths are provided if the patient fails to breath. Arrows indicate patient triggering of breaths.

Modes of Positive Pressure Ventilation

No doubt some physician apprehension concerning mechanical ventilation has been generated by the proliferation of new modes and their confusing acronyms. It is encouraging that in most cases the time honored ventilatory modes provide the best support for our patients. Modes of positive pressure ventilation can be subdivided into two types: volume targeted (controlled) and pressure targeted (controlled). With volume targeted modes, a specified tidal volume (V_T) is delivered and airway pressures vary as changes in total thoracic compliance or airways resistance occur. The advantage of volume targeted ventilation is the guarantee of a minimum minute ventilation (minute ventilation = rate $\times$ V_T) to the patient. This prevents profound hypoventilation even if compliance decreases, airways resistance increases, or respiratory drive is reduced. A disadvantage of volume targeted modes is that airway pressures can become excessive with resultant lung injury if compliance is too low or airway resistance too high. Prevention of occasional marked elevations in airway pressure is provided by the peak pressure setting; if the airway pressure exceeds this preset limit an alarm sounds and further delivery of the breath is terminated. Volume targeted modes are usually employed in adult mechanical ventilation and are the focus of this discussion.

In pressure targeted ventilation, a specified pressure is delivered and V_T and minute ventilation vary with changes in compliance and airways resistance. Pressure targeted ventilation is used for neonates and children and, occasionally, for adults with refractory hypoxemia or diseases causing very low compliance or high airway resistance where the use of volume targeted modes would result in dangerously high airway pressures.

The basic modes of volume ventilation are control mode, assist-control mode (also called assisted mechanical ventilation or AMV), intermittent mandatory ventilation (IMV), and synchronized intermittent mandatory ventilation (SIMV). Recordings of proximal airway pressure are useful in understanding these modes and are shown in Figures 1 and 2. The original form of volume ventilation was control mode, where V_T and respiratory rate were totally regulated by the machine. The patient's inability to alter the respiratory pattern made control mode very uncomfortable for alert patients, necessitating heavy sedation and sometimes neuromuscular blockade.

Assist-control mode has replaced control mode on most modern ventilators because of the improved patient-ventilator interaction that results by allowing the patient to increase the respiratory rate. In assist-control mode the V_T and a baseline rate are preset, but the ventilator senses the patient's additional respiratory efforts and provides *assisted* breaths at the preset V_T.

In IMV, the V_T and a baseline rate are preset, but the patient is allowed to take *unassisted* breaths between the mechanical breaths delivered by the ventilator. Synchronized IMV (SIMV) is like IMV except that the mechanical breaths are provided when the machine senses the patient's initiation of inspiration (Fig. 2). On most recently designed mechanical ventilators, IMV has been replaced by SIMV. Unfortunately, the work of breathing is higher with SIMV than with IMV because the patient must open a "demand valve" to receive flow of gas for each spontaneous breath. The additional work of the patient can be consider-

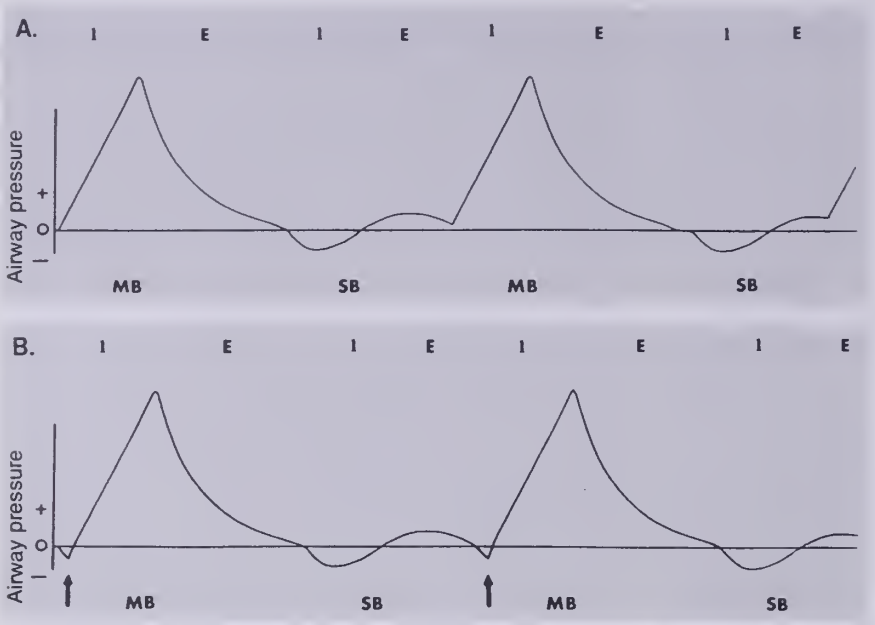


Figure 2. Airway pressure versus time wave forms for IMV (A) and SIMV (B). SB indicates spontaneous breaths and MB indicates mechanical breaths. With IMV, mechanical breaths are not synchronized to spontaneous respiratory efforts. With SIMV, mechanical breaths are triggered by patient inspiratory efforts as indicated by arrows.

able in SIMV when low rates of mechanical breaths are provided (< 6/min.). When using ventilators equipped with *pressure support* mode, the addition of 5 to 10 cm of H_2O of pressure support can considerably reduce the increased work of breathing imposed on the patient by SIMV.[3] As with assist-control, in SIMV the preset rate of breaths is provided by the ventilator even if the patient's respiratory efforts cease.

Both assist control and SIMV (rate of 10-12/min.) are accepted methods of providing full ventilatory support. Proponents of each mode have cited the advantages of SIMV over assist-control[4] and vice versa;[5] however, no well-conducted prospective comparison of the two modes exists. Assist control may be preferable to SIMV for critically ill patients, as it provides a greater capacity for respiratory compensation to normalize pH in the event of a metabolic acidosis.[6]

Basic Ventilator Settings

Although exceptional situations may be encountered, some recommendations can be made for *initial* ventilator settings that are applicable to most adult patients (Table 4). Periodic adjustments in these settings will be necessary and should be based on arterial blood gas measurements and pulse oximetry. The initial ventilatory mode should be assist-control or SIMV. The V_T should be 10 to 15 ml/kg (ideal body weight), precluding the need for sighs. Patients suffering from ARDS have a reduced number of functional lung units and the V_T should be reduced to lessen airway pressures. Tidal volumes as low as 6 ml/kg have been recommended for ARDS patients by some authorities.[7] The usual respiratory rate is 10 to 12 per minute. Patients with severe metabolic acidosis require higher rates to achieve an acceptable pH by respiratory compensation. ARDS patients managed with low tidal volumes are also ventilated with higher rates. Since hypoxemia may develop during the process of intubation, patients should initially be ventilated with 100% oxygen, with subsequent reductions based on arterial blood gas measurements or pulse oximetry.

Inspiratory flow rates are ordinarily set at 60-90 l/min. A good rule of thumb is to set the inspiratory flow rate at four times the patient's expired minute ventilation.[6] Higher flow rates (80-100 l/min) in patients with COPD allow for longer expiratory times and improve oxygenation.[8] Ventilators initiate breaths based on their "sensitivity" to patient inspiratory efforts. A high trigger sensitivity setting lessens the effort required to initiate a breath, but the ventilator may "autocycle," or trigger a breath in the absence of patient respiratory effort, if the sensitivity is too high. Initial sensitivity settings should be high (0.5-1.0 cm H_2O) and reduced (1.0-2.0 cm H_2O) if autocycling occurs. Inappropriately low inspiratory flow rates and low trigger sensitivities can lead to tremendous increases in the work of breathing.[9,10] Adjustment in the sensitivity and inspiratory flow settings of agitated patients often improves patient-ventilator interaction, mitigating the need for patient sedation.

Appropriate Use of Positive End Expiratory Pressure

One of the most controversial aspects of mechanical ventilation involves the application of positive end expiratory pressure (PEEP). The ideal candidate for PEEP suffers from diffuse lung disease with reduced lung volumes (functional residual capacity) and hypoxemia secondary to right-to-left shunt. Prime examples are patients with ARDS or with hydrostatic pulmonary edema from left ventricular failure. In this context PEEP increases functional residual capacity resulting in the recruitment of nonventilated alveoli. This improves pulmonary compliance and lessens shunt leading to improvements in oxygenation.[11] Although PEEP does not reduce *total* extravascular lung water, further improvements in gas exchange occur as PEEP *redistributes* fluid from the alveolar spaces to the interstitium.[12] Patients with unilateral or in homogeneous lung disease are not good candidates for PEEP, as the increased alveolar pressure from PEEP in well-ventilated lung units may result in the shunting of blood to poorly ventilated units, worsening hypoxemia and increasing dead space. Nevertheless, a

Table 4. Initial Settings for Mechanical Ventilation of Adults

	Obstructive Disease	Adult Respiratory Distress Syndrome	Other Patients
Ventilatory Mode	Assist Control (or SIMV)	Assist Control	Assist Control (or SIMV)
Rate (f)	10/min	15-25/min	10-12/min
Fraction of Inspired Oxygen (F_IO_2)	1.0	1.0	1.0
Inspiratory Flow Rate	80-100 1/min	60-90 1/min	60-90 1/min
Tidal Volume (V_T)	10-15 ml/kg	6-10 ml/kg	10-15 ml/kg

These general guidelines may not apply to exceptional patients. Arterial blood gases should be measured 20 minutes after initiation of mechanical ventilation and f, V_T and F_IO_2 adjustments made based on results. V_T recommendations are based on lean body mass.

carefully monitored trial of PEEP can be attempted in these patients when hypoxemia is refractory to high concentrations of oxygen.

The cautious use of low level PEEP (3-5 cm H_2O) has been recommended for selected patients with COPD who require mechanical ventilation.[13] Due to airway obstruction, these patients' lungs may have inadequate time for complete emptying during exhalation, with the resultant development of intrinsic (auto) PEEP. The application of PEEP by the ventilator may improve pulmonary mechanics and reduce the work of breathing of these patients.[14] Caution should be exercised in utilizing PEEP in patients with COPD, as the addition of PEEP occasionally results in an increase in peak airway pressure. If airway pressures rise as PEEP is added, then the PEEP should be discontinued.[13]

The routine use of low levels of PEEP (3-5 cm H_2O) in hemodynamically stable patients has been recommended by some as being "physiologic," or as being useful in compensating for the natural reductions in lung volume that occur in recumbent patients.[15] However, this remains controversial and other authorities recommend against this routine usage of PEEP.[1]

PEEP can result in several untoward effects. Cardiac output may decline due to a reduced venous return to the heart (reduced preload) and hypotension may result. The reduced cardiac output due to PEEP may diminish tissue oxygen delivery despite PEEP-induced increases in the P_aO_2 and arterial oxygen saturation. The risk of barotrauma may increase with PEEP and intracranial pressures may also increase. Alterations in hepatic function (due to impaired venous return) and renal function have been ascribed to the use of PEEP.

When PEEP is employed for hypoxemia it should be used in a systematic fashion. Increases and decreases in PEEP levels should be made in 3-5 cm H_2O increments with ABGs measured 30 minutes later to monitor the effect. The "best PEEP" is the *least* PEEP required to maintain an adequate arterial oxygen saturation ($\approx$90%) using a "safe" F_IO_2 (<0.6) while avoiding significant reductions in cardiac output.[1,15]

PEEP levels greater than 10 cm H_2O are best managed by physicians experienced in the use of mechanical ventilation. When using PEEP values of this magnitude, consideration should be given to the insertion of a pulmonary artery catheter to monitor for PEEP-related effects on cardiac output and oxygen transport.

Summary

The increased complexity of recent mechanical ventilators and the proliferation of new ventilatory modes can be intimidating to the busy practitioner who does not manage mechanical ventilators on a daily basis. However, the clinician can provide good ventilatory support to most patients by familiarity with the indications for, and basic management strategies of, mechanical ventilation.

Acknowledgments

The author is indebted to Mrs. Brenda Callahan for typing the manuscript and to Gary Kinasewitz, MD, for helpful comments during its preparation.

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The Author

Brent Reiff Brown, MD, is an assistant professor of medicine at the University of Oklahoma College of Medicine in Oklahoma City.

SCIENTIFIC

Autologous Bone Marrow Transplantation for Non-Hodgkin Lymphoma

R.A. Saez, MD; R.B. Slease, MD; G.B. Selby, MD; C. Puckett, MD; R.B. Epstein, MD; R.A. Mandanas, MD; D.L. Confer, MD

Non-Hodgkin lymphomas (NHLS) are a group of malignant disorders that can be cured with chemotherapy and/or radiotherapy in 30% to 50% of cases. For those who fail initial therapy, cure is rarely achieved with standard dose chemotherapy; therefore higher doses of chemotherapy have been used with autologous bone marrow support. This major medical center has performed 74 autologous bone marrow transplants (ABMT) for patients with non-Hodgkin lymphoma who had failed initial therapy between 1984 and 1993. Preparatory regimens included high doses of chemotherapy with or without radiotherapy.

There were 14 patients with low grade, 41 with intermediate grade, and 18 with high grade histologies. Among patients with low grade histologies, 90% responded and 50% are relapse-free between 1 and 33 months post-ABMT. Among patients with intermediate and high grade histologies, 25% are relapse-free between 2 and 80 months post-ABMT.

Conclusion: Autologous bone marrow transplantation is effective in patients with relapsed non-Hodgkin lymphoma and should be considered an important therapeutic option.

From the University of Oklahoma Health Sciences Center, Oklahoma City, Okla., and the Medical Arts Pavilion, Newark, Del.

Direct correspondence to Ruben A. Saez, MD, University of Oklahoma Health Sciences Center, Post Office Box 26901, Oklahoma City, Oklahoma 73190

NON-HODGKIN LYMPHOMAS are a heterogeneous group of malignant disorders characterized by high responses to chemotherapy and radiotherapy, leading to cure in 30% to 50% of patients.[1,2] The currently accepted working formulation classifies lymphomas into low, intermediate, and high grade.[3,4] Patients with low grade histologies have a long natural history and typically respond to multiple treatment modalities but are rarely cured. Those with intermediate grade histologies usually require combination chemotherapy at diagnosis; 50% to 80% achieve complete remissions and 35% are cured. Unfortunately, patients who fail to achieve complete remission and those who relapse are rarely cured by salvage regimens.[5,6] High grade lymphomas occur in the very young and in the elderly, have a rapid growth rate, and a predilection for central nervous system involvement. While children with high grade histologies are curable with intensive chemotherapy, adults are rarely curable unless the disease is localized.[7,8]

Bone marrow transplantation allows the delivery of high doses of chemoradiotherapy needed to circumvent tumor resistance by ameliorating bone marrow toxicity with the infusion of hematopoietic stem cells.[9,10] By using the patient's own bone marrow (autologous bone marrow transplantation — ABMT), three- to ten-fold escalation of conventional dosing is possible with limiting toxicities being non-marrow organ damage. The use of a donated bone marrow (allogeneic bone marrow transplantation) is limited by the availability of an HLA-identical sibling or suitable matched unrelated donor, and by patient age due to significant morbidity and mortality secondary to graft-versus-host disease.[11,12]

Since 1984 the bone marrow transplant team at this center has performed 74 autologous bone marrow transplants for non-Hodgkin lymphoma. This paper details the results and future plans.

Patients and Methods

At the center between 1984 and 1993, 74 transplants have been performed in consecutive phase II trials evaluating ABMT in lymphoid malignancies. A total of 73 patients with non-Hodgkin lymphoma were enrolled. Informed consent

Table 1. Pre-Transplant Clinical Characteristics in 73 Patients Receiving Autologous Transplants

Age (years)	
Median	39
Range	4-62
Sex	
Male	39
Female	34
Performance Status*	
Median (range)	1 (0-2)
Prior Therapy	
Chemotherapy	55
Chemotherapy & Radiation	18
Prior Chemotherapeutic Regimens	
Median Number (range)	2 (1-4)
Disease Status Pre-AMBT**	
Untreated	4
Sensitive	42
Resistant	28

*Performance status is defined from 0 for patients fully ambulatory to 4 for fully bedridden

**Sensitive disease means a ≥50% response to chemotherapy at relapse, resistant means a lesser degree of response to chemotherapy or tumor progression, four patients did not receive chemotherapy at relapse and are classified as untreated.

Table 2. Histologic Subtypes According to the Non-Hodgkin Lymphoma Working Formulation

	N
Low Grade	
Diffuse, Small Lymphocytic	2
Follicular, Small Cleaved Cell	10
Follicular, Mixed Small Cleaved and Large Cell	2
Intermediate-Grade*	
Follicular, Large Cell	2
Diffuse, Small Cleaved Cell	2
Diffuse, Mixed Small and Large Cell	7
Diffuse, Large Cell	30
High-Grade**	
Large Cell, Immunoblastic	7
Lymphoblastic	3
Small Non-Cleaved Cell (Burkitt and Non-Burkitt)	8

*Five patients had transformed from Low Grade histology

**One patient had transformed from Low Grade histology

was obtained according to Institutional Review Board guidelines. Forty-three transplants were done with total body irradiation (TBI)- containing regimens (21 with cyclophosphamide and TBI; 21 with cyclophosphamide, etoposide, and TBI; and 1 with cyclophosphamide, BCNU, Ara-C, and TBI), and 31 with chemotherapy-based regimens (17 with cyclophosphamide, BCNU, and etoposide; 7 with cyclophosphamide, carboplatin, and etoposide; 2 with cyclophosphamide, BCNU, etoposide and cisplatin; 2 with cyclophosphamide, etoposide, and cisplatin; 2 with cyclophosphamide and busulfan; and 1 with cyclophosphamide, busulfan, and etoposide.).

Pre-transplantation clinical characteristics are listed in Table 1. There were 39 males and 34 females, ages 4 to 62 years (median age 39 years). Patients had good performance status; they had received a median of two chemotherapy regimens (range 1 to 4) and one-quarter had also received radiotherapy. There were 14 patients with low grade histologies, 41 with intermediate grade histologies, and 18 with high grade histologies (Table 2). Six patients had transformed from low grade to intermediate (5) or high grade (1) histologies. All but one patient received combination chemotherapy at the time of relapse (Table 1). Those patients achieving greater than or equal to 50% reduction in tumor size are classified as having chemotherapy-sensitive disease. Those patients whose disease progressed on their initial treatment or who achieved less than 50% reduction with salvage treatment are classified as having chemotherapy-resistant disease.

Of the 14 transplants for low grade histologies, 11 had chemotherapy-sensitive disease, 2 had resistant disease, and 1 was transplanted in an untreated relapse; the median time from diagnosis to transplant was 25 months (range 12-184 months).

Of the 60 transplants for intermediate and high grade histologies, 31 had chemotherapy-sensitive disease, 26 had resistant disease, and 3 transplants were done in untreated relapse. Thirty-four transplants were done more than one year from diagnosis, while 26 were done within one year. Thirty-three of these 60 patients had an elevated serum LDH immediately prior to transplant. In terms of tumor size, 23 patients had minimal disease (14 in CR and 9 with all nodal masses <2 cm), while 37 had nodal masses ≥2 cm.

Eligibility criteria for autologous bone marrow transplantation included a bone marrow histologically free of lymphoma at the time of harvest, as well as adequate cardiopulmonary, hepatic, and renal function. In five patients, however, peripheral blood stem cells were collected because of a poor marrow harvest (1) or marrow involvement with lymphoma (4).

All patients were managed in private rooms with mask and hand washing isolation. High dose chemoradiotherapy was administered according to the regimens outlined above, and autologous bone marrow and/or peripheral blood stem cells were infused through a central venous catheter on Day 0. Infection prophylaxis was not standardized but most patients received trimethoprim-sulfamethoxazole to prevent pneumocystis, quinalones for antibacterial prophylaxis, acyclovir for positive herpes simplex serology and, since 1991, fluconazole for antifungal prophylaxis. The approach to febrile neutropenia was standardized with broad spectrum antibiotics and amphotericin B for persistent fever. Most patients received total parenteral nutrition and blood component support. Blood products were leukocyte-reduced by filtration and irradiated. Of the 69 transplants involving autologous bone marrow, 28 received hematopoietic growth factors (GM-CSF,

factors (GM-CSF, G-CSF, and IL-3) following marrow infusion.

After hematologic recovery, full restaging was performed; irradiation of residual disease was done if possible. Complete remission was defined as disappearance of all tumor by physical examination and noninvasive studies; partial remission required at least 50% reduction in measurable lesions.

Life table estimates of relapse-free survival and overall survival were calculated by the method of Kaplan and Meier,[13] measured from the date of ABMT to the date of progression or death.

Table 3. Engraftment Parameters in 74 Autologous Transplants for Non-Hodgkin Lymphoma

	PBSC*	PBSC +BM	BM†	BM +HGF‡
Number of transplants	4	1	41	28
Mean NC** x 10^8/kg	10.0	8.5	2.42	2.39
(range)	(4.3-21.3)	(0.7)	(1.11-5.49)	(1.1-4.76)
Median days to ANC†† ≥ 500/μl	15	11	16	15
(range)	(14-46)		(11-40)	(7-38)
Median days to platelets ≥20,000/μl	18	NE†††	21	22
(range)	(13-48)		(10-120)	(13-70)

* PBSC, peripheral blood stem cell infusion
† BM, bone-marrow infusion
‡ HGF, hematopoietic growth factor support (G-CSF, GM-CSF and/or Interleukin-3)
** NC, nucleated cells
†† ANC, absolute neutrophil count
††† NE, not evaluable

Results

Engraftment.— As seen in Table 3, a mean of 2.4 × 10^8 nucleated marrow cells/kg (range 1.1-5.49) were infused in 70 patients. 9.7 × 10^8 nucleated peripheral blood cells/kg were infused in five patients in whom adequate marrow could not be obtained. In patients receiving bone marrow only, the addition of growth factors did not hasten neutrophil (15 vs. 16 days to 500 neutrophils per μl, respectively) or platelet (22 vs. 21 days to 20,000 platelets per μl, respectively) recovery. The use of peripheral blood stem cells in patients with unsuitable marrows for harvesting achieved similar neutrophil recovery and perhaps slightly earlier platelet recovery when compared with autologous marrow.

Toxicity.— A detailed analysis on toxicity is beyond the scope of this report, but clearly the TBI-containing regimens were more toxic than chemotherapy-only regimens. TBI-containing regimens (particularly in combination with etoposide) had as major toxicity diffuse alveolar hemorrhage, BCNU-based regimens had higher incidence of venoocclusive disease of the liver, while etoposide increased the severity of oral mucositis.

There were 11 regimen-related deaths (15%) and major contributing causes were: diffuse alveolar hemorrhage (4), encephalopathy (2), bacterial (1) or fungal (1) sepsis, congestive heart failure (2), and venocclusive disease of the liver (1). The cyclophosphamide/etoposide/TBI had the highest rate of toxic death (5 of 21 patients, 24%), while there have been no toxic deaths among 20 patients using our most recent regimen, cyclophosphamide/ etoposide/BCNU. There have been four late deaths in patients without tumor progression due to idiopathic pneumonitis (3 and 10 months), fungal pneumonia (10 months) and lung cancer (23 months).

Tumor Response.— Overall, 22 of 73 NHL patients remain alive and free of disease with a median follow-up as of February 1994 of 20 months (2-80 months). Two patients are alive with recurrent disease following relapse (3 and 5 months). The 3-year progression free survival is 23% (Fig. 1).

There are no survival differences between TBI and non-TBI containing regimens. It is unknown whether our most recent regimens (cyclophosphamide/TBI/ etoposide and cyclophosphamide/ BCNU/etoposide) have had an impact in survival since the follow-up is too short. In Figure 2, the bone marrow transplant outcome according to histologic grade is presented.

Low Grade Lymphomas.— The characteristics and outcome of 14 patients with low grade histology are detailed in Table 4. The response rate was 90%, and 50% are free from progression at a median of 12 months (range 1 to 33 months) from transplantation. Ten of 14 patients (71%) had a history of bone marrow involvement prior to bone marrow harvest (6) or peripheral blood stem cell collection (4), but this did not

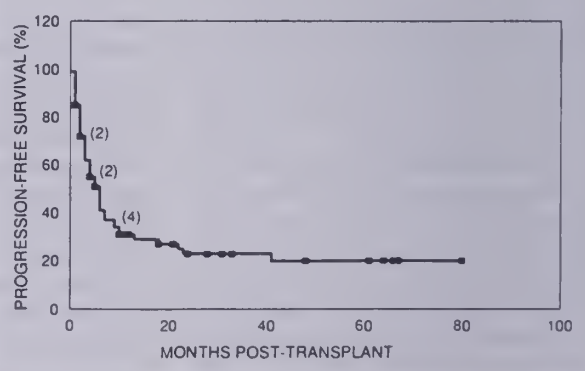

Figure 1. Outcome following autologous bone marrow transplantation for non-Hodgkin lymphoma; progression free survival curve includes two patients with residual radiographic abnormalities of uncertain significance. Each symbol (■) denotes a patient who is alive and progression free; numbers in parenthesis indicate multiple survivors at a single time point.

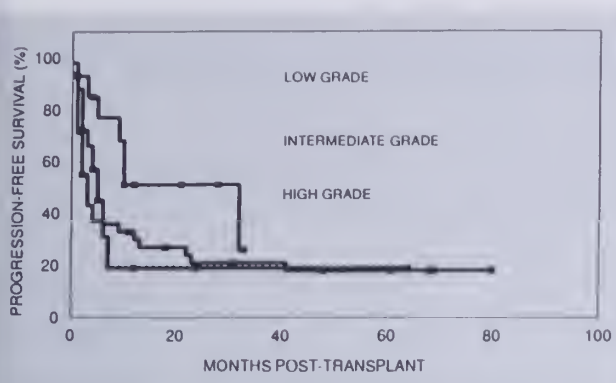

Figure 2. Outcome following autologous bone marrow transplantation for non-Hodgkin lymphoma by grade; top curve low grade, middle curve intermediate grade and bottom curve high grade. Each symbol (■) denotes a patient who is alive and progression free; numbers in parenthesis indicate multiple survivors at a single time point.

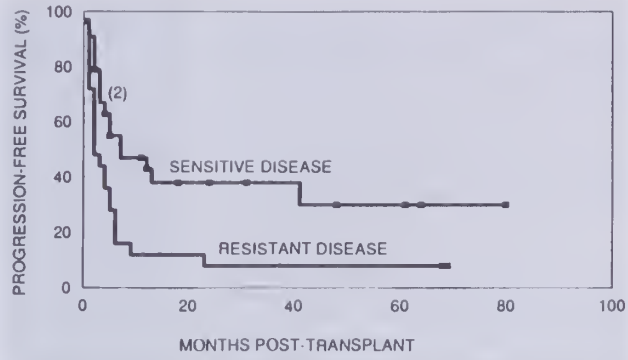

Figure 3. Outcome following autologous bone marrow transplantation in non-Hodgkin lymphoma by disease status pre-transplant, sensitive vs. resistant. Each symbol (■) denotes a patient who is alive and progression free; numbers in parenthesis indicate multiple survivors at a single time point.

appear to adversely affect outcome post-ABMT (data not shown). Of note, 3 of the 4 patients rescued with peripheral blood stem cells remain in complete remission, but their follow-up is short.

Intermediate–High Grade Lymphomas.— Overall, 15 of 59 patients (25%) with intermediate or high grade histology are free from progression at a median of 24 months (range 2 to 80 months). In analyzing our data, we examined several prognostic factors determined by others to influence outcome (Table 5).[14] Among patients whose tumors were chemosensitive at the time of relapse (sensitive relapse), 39% are free from progression, while this was a rare event (8%) in those with resistant disease (Fig. 3). We also observed a trend toward significance for tumor size and LDH level (Table 5). These factors are interrelated; i.e., those in sensitive relapse were more likely to achieve minimal disease status and to have a normal serum LDH pre-ABMT.

Neither time from diagnosis to transplantation nor the histologic subgroup had prognostic significance (Table 5). The subgroup of patients who were transplanted within one year of diagnosis because of high risk factors (but prior to relapse) had the best outcome with 50% freedom from progression. Radiotherapy was administered to 13 of 22 patients with residual tumor masses post-ABMT. Patients who received consolidation radiotherapy had a long-term outcome similar to those who were in complete remission post-ABMT (38% vs. 42% freedom from progression, p=NS) and superior to those with residual masses who did not receive radiotherapy (data not shown).

Table 4. Disease Characteristics and Response to Transplantation in 14 Patients with Low Grade Non-Hodgkin Lymphoma

N	14
Median Age (range)	45 (33-59)
Disease Status	
▶ CR1/PR1	2
▶ CR2/PR2	5
▶ ≥CR3/PR3	4
▶ Refractory first relapse	2
▶ Untreated first relapse	1
Median Time from Diagnosis to BMT (range, mos)	25 (12-184)
BMT Regimen	
▶ TBI	8
▶ Non-TBI	6
Outcome	
▶ Response rate (%)	90
▶ Free from progression	7
▶ Duration of response (range, mos)	1-33+

CR–Complete Remission; PR–Partial Remission; BMT–Bone Marrow Transplant; TBI–Total Body Irradiation

Discussion

Over the last 20 years, advances in combination chemotherapy and radiotherapy have led to the cure of many patients with non-Hodgkin lymphoma. Unfortunately, those who do not achieve a complete remission and those who relapse have a dismal outcome with conventional therapy. With the advent of autologous bone marrow transplantation it has become feasible to utilize increasing doses of chemoradiation in the treatment of these tumors.[15] In the last 10 years the bone marrow transplantation team has performed 74 autologous bone marrow and/or peripheral blood stem cell transplants for patients with non-Hodgkin lymphoma who failed to achieve a complete response to induction therapy or who relapsed after an initial response. The results as of February 1994 indicate that 30% of these patients are free from progression. These results are similar to those achieved by others.[16-18]

In the last five years we have tried to improve the results achieved with the standard cyclophosphamide/TBI regimen by adding new drugs (etoposide) or substituting chemotherapy (BCNU, etoposide) for total body irradiation. While the follow-up is too short to assess whether the newer regimens have greater antitumor effect, we have found the cyclophosphamide/TBI/etoposide regimen to be too toxic (24% mortality) due to diffuse alveolar hemorrhage. The current chemotherapy-based regimen (cyclophosphamide/BCNU/etoposide) has a high response rate (94%) with no treatment related mortality.

Since non-Hodgkin lymphoma is a very heterogeneous disease, we have analyzed the results according to histologic categories. The patients with low grade lymphoma had a high response rate and 50% remain in remission at a median of 12 months. Because the biology of these lymphomas is associated with late relapses, a longer follow-up is needed to evaluate the true impact of ABMT. Because all these patients have bone marrow involvement when evaluated by molecular techniques, several groups have advocated the use of purging and/or peripheral blood stem cells.[19,20] Our experience with the latter is encouraging and we plan it to be a focus of our research.

In contrast to the low grade histologies, patients with relapsed intermediate and high grade lymphomas treated with salvage chemotherapy have <10% survival at 3 years.[21] Of our 59 patients with intermediate or high grade histology, 25% remain free from progression at a median of 24 months. Thus, our results and long-term follow-up suggest cure of disease for a portion of patients in this population. Unfortunately, the remaining patients relapse, usually at sites of previous involvement. We have identified the chemosensitivity of the tumor to chemotherapy as the most important predictor of good outcome: those who achieve a complete or partial response prior to transplant have a 39% freedom from progression, vs. 8% for non-responders (p<0.01). Sensitivity to chemotherapy is associated with lower tumor bulk (size <2 cm) and normal LDH, factors which also proved predictive in our analysis.

Table 5. Prognostic Factors Prior to Autologous Bone Marrow Transplantation in Intermediate and High Grade Non-Hodgkin Lymphoma

Better				Worse		
Factor	n	FFP (%)*	P+	FFP (%)	n	Factor
Sensitive relapse	31	12 (39)	<0.01	2 (8)	25	Resistant relapse
Tumor <2cm	23	9 (39)	<0.1	7 (19)	37	Tumor ≥2cm
LDH normal	27	9 (33)	<0.25	6 (18)	33	LDH elevated
>12 mos from diagnosis	34	8 (24)	>0.25	2 (13)	16	<12 mos from diagnosis
Intermediate grade histology	42	11 (26)	>0.25	4 (22)	18	High grade histology

*FFP, Freedom From Progression
+p, Significance by Chi-Square

How to improve our results? Because patients who are resistant to salvage chemotherapy do poorly, we should identify high risk patients prior to relapse. In this report, 10 patients were transplanted in first CR/PR because of high risk features and 5 of these (50%) remain in remission. Similarly, investigators have identified factors at diagnosis that predict for poor outcome: poor performance status, high LDH, stage III-IV, and tumor bulk (nodal masses ≥ 10 cm or ≥ 2 extranodal sites).[22] At our center we have a protocol involving induction chemotherapy followed by ABMT for newly diagnosed patients with intermediate-grade lymphomas and high risk features.

Table 6 illustrates our current approach to patients with non-Hodgkin lymphoma. Since patients with resistant disease do poorly, only rarely will these patients be candidates for marrow transplantation. Peripheral blood stem cells will be used for all patients with low grade lymphoma and for patients with intermediate-high grade lymphoma with marrow involvement. Allogeneic bone marrow transplantation will be used in young patients (<50 years old) with marrow involvement and/or lymphoblastic histology who have an HLA-identical sibling.

Table 6. Indications for Autologous Bone Marrow Transplant for Non-Hodgkin Lymphoma

1. High-Grade Lymphoma
   - —Stage II (≥ 10cm), Stage III-IV, after induction chemotherapy response
   - — Sensitive Relapse
2. Intermediate-Grade Lymphoma
   - — High risk features at diagnosis (protocol enrollment at diagnosis)
   - — Sensitive Relapse
3. Low-Grade Lymphoma
   - — Induction Failure
   - — Sensitive Relapse

In summary, our experience over the last 10 years suggests that high dose chemotherapy can cure some patients with relapsed non-Hodgkin lymphoma. The major impediment is tumor recurrence, so our focus will be on strategies to decrease relapses.

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The Authors

Ruben A. Saez, MD, is assistant professor of medicine and associate director of bone marrow transplantation at the University of Oklahoma Health Sciences Center in Oklahoma City.

R. Bradley Slease, MD, is clinical professor of medicine at Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pa.

George B. Selby, MD, is associate professor of medicine at OUHSC and assistant chief of the Medical Service at Veterans Administration Medical Center of Oklahoma.

C. Puckett, MD, is medical oncologist at Medical Arts Pavillion, Newark, Del.

Robert B. Epstein, MD, is Eason Professor of Medicine at OUHSC.

Romeo A. Mandanas, MD, is assistant professor of medicine at OUHSC.

Dennis L. Confer, MD is associate professor of medicine and director of marrow transplantation at OUHSC, and medical director of the National Marrow Donor Program, Minneapolis, Minn.

SCIENTIFIC

Treatment of Putative Non-A, Non-B, Non-C Hepatitis with α Interferon: A Preliminary Trial

D. H. Van Thiel, MD; J. S. Gavaler, PhD; Nahed Baddour, MD; Lois Friedlander, BSc; H. I. Wright, MD

Chronic hepatitis due to putative non-A, non-B, non-C hepatitis occurring in an individual who is negative for HBV and HCV markers has been identifiable only recently. Little or nothing is known about its natural history or response to interferon therapy. In the present study, 13 subjects with chronic non-A, non-B, non-C hepatitis were treated with interferon for 6 months (5 million units, three times per week). Prior to and after 6 months of therapy and again 6 weeks after discontinuing interferon therapy, each subject underwent a liver biopsy. These tissues were used to define the histopathology, the character of the cellular infiltrate within the liver, and the changes in histopathology and inflammatory infiltrate achieved in response to interferon therapy and withdrawal. No differences for age, gender, initial AST, bilirubin, histopathology, or Knodell score were evident between responders (n=7) and non-responders (n=6). Only the number of NK cells was altered significantly as a result of IFN treatment and distinguished responders from non-responders.

These data demonstrate that: (1) chronic non-A, non-B, non-hepatitis can be treated with interferon; (2) interferon activates NK cells and enhances hepatocyte expression of Class II MHC antigens; and (3) interferon also increases the number of CD3, CD4, and CD8 cells found within the liver but these changes do not distinguish between responders and non-responders.

CHRONIC HEPATITIS is well recognized as a significant health hazard which often leads to cirrhosis and its various complications, particularly those associated with portal hypertension and the development of hepatocellular carcinoma.[1,2] Until recently, most cases of chronic hepatitis were thought to be the result of a chronic viral infection due to the hepatitis B virus (HBV). With the development of HBV serologic tests and more recently polymerase chain reaction (PCR) techniques, many cases of putative chronic viral hepatitis have been found not to be a result of a chronic HBV infection.[3-10]

The recent recognition of hepatitis C (HCV) as a distinct viral agent, which is the major but not sole cause of chronic viral hepatitis and hepatocellular carcinoma not due to HBV, has lead to the recognition that yet another type of chronic viral hepatitis must also exist. This latter form of viral hepatitis has, for lack of a better name, been termed as non-A, non-B, non-C hepatitis (NANBNC) by exclusion of the other better known and fully characterized viruses which cause chronic viral hepatitis.[1,2] Because the recognition of NANBNC hepatitis has only occurred recently, little is known of its natural history. For example, although it is recognized as a cause of chronic viral hepatitis, whether it can progress with any frequency to cirrhosis and whether it is a cause of hepatocellular carcinoma are as yet unknown. Nonetheless, the disease can be identified, at least tentatively, by exclusion of HBV and HCV as well as other recognized causes of viral hepatitis such as hepatitis A, cytomegalovirus, Epstein Barr virus, and other less well known

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

but identifiable viruses using a combination of serologic and molecular biology testing procedures, especially polymerase chain reaction (PCR).

Interferon α 2B (IFN) has been used as an effective agent in the treatment of chronic viral hepatitis.[11-40] For several years, it has been used for the treatment of HBV.[25-41] Clinically, it has been used widely and is currently FDA-approved as the only available treatment for HCV disease.[11-24] The role of IFN therapy in the treatment of chronic hepatitis NANBNC is currently unknown. To determine its value in the clinical management of individuals with putative chronic NANBNC hepatitis and to determine its effect upon the type of cells comprising the cellular infiltrate in this disease, the following study was performed.

Table 1. Characteristics of the IFN Responders and Non-Responders

	Responders	Non-Responders
Number (n)	7*	6
Gender (m/f)	3/4	4/2
Age Years	39.1 ± 3.4	41.5 ± 6.2
AST (iu/l)	172 ± 32	131 ± 37
Bilirubin (mg/dl)	1.0 ± 0.1	0.9 ± 0.1
Histopathology		
CPH	3	4
CAH	1	1
CAH & Cirrhosis	3	1

*2 full responders and 5 partial responders

Materials and Methods

Subjects.— Thirteen subjects with chronic viral hepatitis defined by the combination of a minimum of 6 months of abnormal serum transaminase levels, a liver biopsy consistent with chronic viral hepatitis, and no serologic or histologic evidence for HBV, HCV, Wilson's disease, α_1 antitrypsin deficiency, hemochromatosis, CMV, or EBV disease were identified and agreed to participate in this study. All 13 had had one or more possible parenteral exposures either in terms of a blood transfusion or the use of intravenous drugs. At enrollment, each underwent a biochemical and serologic examination as well as a percutaneous liver biopsy with tissue cores being obtained for routine histologic evaluation, a quantitative determination of the hepatic content of iron and copper, and characterization of the mononuclear cell infiltrate present within the biopsy. A prothrombin time and platelet count were obtained before each liver biopsy. The panel of biochemical and serologic studies obtained on each subject included the following:

- HAV—IgG and IgM antibody
- HBV—HBsAg, HBsAb, HBCAb, HBeAg, HBeAb
- HCV—HCV-Ab (second generation ELISA and recombinant immunoblot [RIBA II])
- HDV—antibody IgG only
- Serum α_1-antitrypsin level and phenotype determinations
- Severe iron, total iron binding capacity and ferritin levels
- Serum ceruloplasmin level
- Serum total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (AP), gamma glutamyltranspeptidase (γ GTP)
- CMV and EBV serologic studies
- HLA testing for A, B, and Dr antigens
- Antinuclear antibody, antismooth muscle antibody, and antimicrosomal antibody

All of the HCV assays were run in duplicate. Each HCV assay, including PCR on the serum using a nested PCR method and primers specific for the 5' end of the virus, was run retrospectively after the interferon therapy had been completed using serum obtained before and after the IFN therapy.

Protocol.— Having completed the above entry assessments, and having tested negative on two occasions separated by 6 months for HCV by PCR and antibody testing (retrospectively as noted above) as well as the presence of antinuclear antibody, antismooth muscle, and antimicrosomal antibody, each subject was treated for 6 months with αIFN 2b at a dose of 5×10^6 units administered subcutaneously 3 × week. The specific product utilized was produced by Schering-Plough Laboratories, New Jersey. Each subject was monitored during the course of the IFN therapy with twice weekly complete blood counts to include a platelet count and serum determinations of total bilirubin, ALT, AST, AP, and γ GTP for two weeks, then weekly for an additional two weeks, then monthly thereafter until completion of a 6-months course of IFN. These studies were performed in a variety of hospital laboratories which serviced the patients in their local communities. Each such laboratory was fully accredited. Each forwarded the results to the investigators as each scheduled assessment was made. At the completion of 6 months of IFN therapy, a second liver biopsy was obtained for follow-up histologic and immunohistochemical studies. Serum biochemical parameters were followed monthly for an additional 6 months. Six weeks after the second biopsy, a third liver biopsy was obtained to confirm the findings noted after 6 months of IFN treatment and to determine whether IFN treatment had caused either a transient worsening and/or a persistent improvement in the histopathology and chronic inflammatory infiltrate present within the liver tissue by comparing the second and third biopsies.

This study was approved in April 1990 by the Institutional Review Board responsible for assessing human studies at the University of Pittsburgh.

Liver Histology.— All liver biopsies for histopathologic evaluation were fixed overnight in 10% buffered formalin and prepared for histologic examination by the Clinical Pathology Department of Presbyterian University Hospital, Pittsburgh, Pa. Each biopsy was sectioned at 5μ and representative sections were stained with hematoxylin and eosin, Masson's trichrome, and a reticulum stain. All biopsies were read and

officially interpreted by staff pathologists at Presbyterian University Hospital who were both not knowledgeable about and were not participating in this study. Each biopsy was subsequently blindly re-read by one of the investigators and assigned a Knodell score based upon established criteria.[21]

Liver Immunohistochemistry.—A core of tissue obtained at each biopsy time point was immediately embedded in OCT medium and prepared for immunohistochemical staining utilizing the avidin-biotin complex (ABC) technique.[22,23] A panel of monoclonal antibodies developed against specific cell surface markers on mononuclear cells was utilized. The specific monoclonal antibodies utilized in this study included OKT3 (pan lymphocyte), OKT4 (helper T cell), OKT8 (suppressor/cytotoxic T cell), GA (natural killer cell), and MHC-Class I antigens and MHC-Class II antigens. With each run with each monoclonal antibody, appropriate positive and negative controls were utilized in order to be confident about the staining results. For each biopsy and each monoclonal antibody, a total of 5 high power fields centered upon the portal areas were counted for the number of positively staining cells. The results for each antibody with each biopsy were recorded as the total value for the 5 portal fields assessed.

Response Definitions.—A full clinical response to IFN therapy was defined as a complete normalization of the serum ALT levels that persisted for 6 months after discontinuing the IFN therapy. A partial response to IFN therapy was defined as a >50% reduction but not a normalization of the serum ALT level for 6 months after discontinuing the IFN therapy. All other responses were defined as a non-response to IFN therapy.

Statistical Analysis.— All results are reported as mean values ± SEM. Comparisons between responders and non-responders for age, entry levels of bilirubin, and AST were made by using the student's T test. ANOVA, utilizing appropriate comparison procedures, was utilized to analyze the results of the various immunoperoxidase studies. A p value <0.05 was considered to be significant.

Results

Of the 13 patients enrolled in this study, 7 were male and 6 were female. Their mean age was 40.2 ± 3.3 years. Seven had histologic chronic persistent hepatitis; 2 had chronic active hepatitis; and 4 had chronic active hepatitis with cirrhosis. This relatively advanced stage of histologic hepatic disease was documented further by a mean five-fold elevation of the serum AST level at entry (153 ± 24 iu/l) and a high normal serum bilirubin level (0.9 ± 0.1 mg/dl). When this group of 13 patients was segregated into those who responded (full and partial responses) and those who did not (failures), no differences between the two groups were identifiable for gender, age, initial AST level, initial serum bilirubin level, or initial histopathology as shown in Table 1. Only two of the responding patients were full responders; five were partial responders despite the fact that full dose therapy was continued throughout the treatment period.

The mean Knodell score of the total group at entry was 7.2 ± 1.6. When this initial score was segregated into those who ultimately responded (8.0 ± 2.7) compared to those who did not (6.2 ± 1.7), no differences were noted between the two groups. Moreover, no differences were noted throughout the subsequent course of the study (Table 2). Improvement in the Knodell score occurred as a result of a reduction in the intensity of the portal inflammatory response as well as hepatocyte necrosis throughout the hepatic lobules.

The cell surface characteristic of the cells found within the liver of these individuals with NANBNC hepatitis is shown in Table 3. The table reports the data collected at each of the 3 times a liver biopsy was obtained. With interferon treatment, the number of CD3+, CD4+, and NK cells within the liver increases. Only the increase in NK cells ($p<0.01$) was significant however. Moreover, the number of CD3+ and CD4+ cells continued to increase despite having discontinued the IFN 6 weeks prior to the time of the third biopsy. In contrast, the number of NK cells returned to the basal pre-IFN level. Of some interest was the finding that little or no change in the expression of Class I or Class II on lymphocytes was noted at each time, although a trend for more positive staining cells was evident with each additional biopsy. Little or no change in the expression of Class I antigens by liver cells was evident throughout the study. In contrast, Class II antigen expression by liver cells increased after 6 months of IFN therapy and then returned to basal pre-IFN levels following discontinuation of the IFN therapy. In contrast to what was seen with hepatic parenchymal cells, bile duct cells showed a steady increase in the number of cells staining positive for Class I and Class II antigens with time and IFN treatment. The meaning of this latter finding is unclear.

Discussion

This is the first comprehensive study of the effect of IFN treatment in the clinical management of individuals who have chronic hepatitis thought to be of viral origin who repeatedly test negative for evidence of prior HBV and HCV infection using PCR, a second generation ELISA, and the RIBA-II assay (all performed retrospectively on stored sera) and, as a result, are classified as having

Table 2. Knodell Scores of the Patients Studied

	At Entry	At 6 Months	At 6 Months+6 Weeks
Total Group	7.2 ± 1.6	7.5 ± 1.2	7.0 ± 1.2
Responders	8.0 ± 2.7	7.7 ± 2.0	5.8 ± 1.8
Non-responders	7.2 ± 1.7	7.4 ± 1.4	8.4 ± 1.6

Table 3. Characteristics of the Mononuclear Infiltrate Present Within the Liver at Each of the Three Time Points Studied

	Entry	6 Months	6 Months+6 Weeks
CD3+	268 ± 56	316 ± 68	376 ± 135
CD4+	168 ± 36	210 ± 53	297 ± 118
CD8	119 ± 27	122 ± 22	105 ± 34
G8+	21 ± 5	30 ± 4	17 ± 5
Class 1+ Lymphocytes	153 ± 39	159 ± 44	278 ± 119
Class 2+ Lymphocytes	193 ± 46	232 ± 57	211 ± 76
Class 1+ Liver Cells	15 ± 3	18 ± 3	21 ± 7
Class 2+ Liver Cells	21± 3	29 ± 2	19 ± 3
Class 1+ Bile Duct Cells	22 ± 4	34 ± 5	46 ± 13
Class 2+ Bile Duct Cells	14 ± 2	22 ± 3	30 ± 4

NANBNC hepatitis. It is recognized by the authors that PCR of liver tissue may be the only definitive way of eliminating the possibility of HCV disease in the patients studied. Nonetheless, the authors strongly believe that such is not the case based upon the following data. First, the patients studied tested negative for HCV using PCR and failed to have identifiable HCV antibody by the best tests available for this determination. Second, the response rate for the subjects studied was different from that experienced by patients with HCV disease treated by the authors. Thirdly, the characteristics of the cellular infiltrate within the liver and its response to IFN therapy differs from that seen in patients with HCV disease.

As has been the case with hepatitis C, a majority of the patients treated with IFN for 6 months in this study experienced a biochemical response with a reduction in their serum ALT levels.[11-24] Unfortunately, as has been the case in other series, when the hepatic histopathology was assessed before and after IFN therapy, no benefit of IFN therapy has been noted.[24] Instead, the post-treatment biopsies appeared either the same or, more often, were worse.

Unlike the situation with HBV, where few responses to IFN treatment are noted,[25-41] but similar to the situation with HCV,[11-24] no evidence for an increased rate of IFN responses was evident for the responder groups based upon gender, age, initial ALT level, or severity of the histopathology at the time of initiation of the IFN treatment.

It is important to note that in this study, the findings of the immunoperoxidase studies and the assessment of the Knodell scores gave similar results although they were completed by different investigators involved in the research and without knowledge of the other investigator's results until the individual assessments were complete. This finding was somewhat unexpected in that based upon the biochemical data available, an improvement (reduction in the inflammatory infiltrate) would have been expected on histologic reassessment rather than a deterioration (increase in the number of infiltrating cells). The only change in the character of the inflammatory infiltrate that mirrored the changes in IFN therapy was the increase in NK cells noted at 6 months and their return to basal levels after the cessation of IFN therapy (Table 3). It may be of some interest that the expression of Class II antigens on liver cells also mirrored the change in the IFN therapy during this study (Table 3).

It is not possible to rule out a role for IFN-induced changes in CD3+ and CD4+ cell number as a means of determining whether or not an IFN response was likely to be observed or not as each of these cell types increased in number within the liver with IFN treatment. The problem in interpreting this change, however, was that despite having discontinued the IFN for 6 weeks, the number of these cell types continued to increase at least until sometime after the third liver biopsy was obtained. Whether this reflects a continued requirement for these cells once having been recruited as a result of earlier IFN therapy or represents a non-specific effect of IFN treatment that persists after its initiation cannot be determined by the data available as part of this investigation. What is apparent is that the changes herein reported for patients with putative hepatitis NANBNC are quite different from those previously reported for individuals with hepatitis B and/or hepatitis C.[42-43]

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The Authors

David H. Van Thiel, MD, is medical director of the transplant program and chief of hepatology disease at Baptist Medical Center (BMC).

J.S. Gavaler, PhD, is director of women's studies at BMC.

Nahed Baddour, MD, is a professor of pathology in Alexandria, Egypt.

Lois Friedlander, BSc, is nurse coordinator, liver disease, at West Penn Hospital in Pittshurgh, Pa.

H.I. Wright, MD, is a staff member, transplantation medicine, BMC.

SPECIAL

Oklahoma's Rural Health Providers and Consumers Prioritizing Rural Health Issues

Gerald A. Doeksen, PhD; Cheryl F. St. Clair, BS; Richard Perry, MA

The Clinton administration has been busy for many months devising a new health care plan to address national needs. Over the last three years, rural health providers and consumers in the State of Oklahoma have also been actively reviewing their state health issues and concerns and determining the priorities. The purpose of this paper is threefold: (1) to illustrate why it is vital to have a strong health care sector in rural Oklahoma; (2) to review the issues and concerns identified by rural Oklahomans; and, (3) to provide a philosophical basis for possible solutions to the barriers identified.

From the Oklahoma Cooperative Extension Service of Oklahoma State University, Stillwater, and Rural Health Projects, Inc., Enid.

Direct correspondence to Gerald A. Doeksen, PhD, Oklahoma State University, Department of Agricultural Economics, 508 Ag Hall, Stillwater, OK 74078.

The Importance of the Rural Health Sector to Rural Communities

Activities such as the Governor's Rural Health Conference and regional meetings sponsored by the Governor's Commission on Health and the Oklahoma Rural Health Association (OKRHA) identified problems, the solutions to which are necessary to making and keeping rural residents healthy. A viable health sector is also needed for the economic health of rural communities. The importance of the health sector on rural development and growth is often overlooked; thus we want to summarize how crucial the health sector is for a healthy community economy.

Research illustrates that a viable health sector is needed in rural communities because (1) the health sector directly and indirectly employs a large number of people; (2) the health sector is important for industrial growth; and (3) the health sector is important for attracting retirees.

The Importance of the Health Sector as an Employer.— It is often said that hospitals are the second largest employer; often, only schools employ more. If we look at all components of the health system, we will find that often 10% of the people are directly involved in health care and another 5% indirectly employed by the health sector. For example, the number of people directly working in the health sector in Perry, Oklahoma, is presented in Table 1. In this small community with a population of 4,987 and employment of 2,113, the number of people working in the health sector is 231. The payroll is estimated at $5,031,468.

As these people spend money, their dollars spread throughout the community and create secondary employment and income impacts. The secondary impacts on the Perry economy are presented in Table 2 with the use of economic multipliers. The bottom line is that the health sector directly and indirectly employs 300 people or 14% of the community's employment. The total payroll of the health sector is estimated at $7,094,369. This sector has a large impact on Perry merchants. It is estimated that $1,915,479 is generated in retail sales and $76,619 in sales tax collections. In summary, the health sector is very important to the economy of a rural community.

The Importance of the Health Sector for Industrial Growth.— As rural communities attempt to diversify their economies, retaining existing businesses and industries are generally growth strategies. The question that arises is how important is a viable health care

Table 1. Direct Ecomonic Activities of the Health Sector in Perry, Oklahoma

Component	Estimated Employees	Estimated Payroll
Hospital (28 beds)	65	$1,561,968
Physicians (6)	24	740,000
Dentists (2)	9	248,000
Optometrists (1.5)	3	133,500
Pharmacies (4)	16	488,000
Nursing Homes (2-153 beds)	100	1,510,000
County Health Department	7	175,000
Home Health Care	7	175,000
Totals	231	$5,031,468

Table 2. Employment, Income, Retail Sales, and Tax Collections Resulting from Perry Health Sector

Item	Health Sector Workers Direct Impact	Total Community Effect Direct and Secondary Impacts	Multiplier
Employment	231	300	1.30
Income	$5,031,468	$7,094,369	1.14
Retail Sales	$1,358,496	$1,915,479	
Sales Tax	$ 54,339	$ 76,619	

sector to business and industry decision-makers as they evaluate a community for location purposes. Research studies investigating this hypothesis are few. One study (Lyne, 1988) found that quality-of-life (QOL) factors are playing a dramatic role in location decisions. The study concluded:

"In fact, almost half (of facility planners) say QOL considerations are controlling both initial screening and final site selections."

The most important QOL variables were transportation, education, and health. A related finding by Lyne (1990) is the role of health care costs in industrial location decisions. Specifically, Lyne's survey of corporate executives indicated that corporations are sometimes giving priority to sites which provide health services at low costs.

In fact, 17% of the respondents indicated their companies are using health care costs as a tie-breaking factor between comparable sites. To the extent rural areas are able to provide health care at a lower cost than their urban counterparts, this development may bode well for some rural areas if quality can be assured as well.

The Importance of the Health Sector for Attracting Retirees.—Retirees form a special group of residents whose spending and purchases can be an important source of local jobs. Additionally, middle and upper income retirees often have substantial net worth. Many rural areas have environments (e.g., good climate and outdoor activities) that enable them to be in a good position to attract retirees. Retaining retirees is, of course, just as important as attracting new retirees, and the rural population contains a relatively high proportion of elderly, including retirees. The amount of spending embodied in this population, including the purchasing power associated with Social Security, Medicare, pensions, and other transfer payments is substantial. Hence, a critical economic development question is the extent to which the availability of health services influences the location decision of retirees.

Although the data are limited, several studies suggest that health services may be a critical variable. For example, Toseland and Rasch (1978) conducted a survey of 878 persons 55 years of age or older in 28 communities in the U.S. The four items that were the best predictors for retirement location were safety, recreation, housing, and health care. Another study by Reginier and Gelwicks (1981) surveyed 221 people 60 years and older who were considering a retirement community. Nearly 60% said health services were in the "must have" category. Only protective services were mentioned more often than health services as a "must have" service. Finally a case study in rural North Carolina (Pomeranz, 1988) noted that the:

"...lack of local long-term services and hospital beds has resulted in increasing numbers of seniors being forced to receive medical care in the same distant locations (fifty miles away or more) as they are hospitalized. This has resulted in a service displacement cycle in which many of these seniors have been forced to relocate in order to receive needed rehabilitation and support services (p. 44)."

What Rural Oklahomans Perceive as Barriers to Quality Health Services Delivery

It is clear that health care is important to a local rural economy. Rural residents know this and have not been afraid of speaking their mind about it. The Area Health Education Centers (AHECs) started the process of sounding out rural opinions on health issues two years ago (Table 3). In 1991 the Oklahoma AHEC system sponsored a series of health forums across the state to ask all Oklahomans what the issues were. Personnel shortages, regulation, and lack of health insurance surfaced as major concerns, along with hospital and professional licensing, public health services, home health care, emergency services, and transportation.

It became apparent that the issue of health care was much broader than regional concerns. A statewide effort would be needed to better define the issues and the priorities of health care, and to determine possible resolutions of the issues. Governor David Walters responded by calling a Rural Health Conference in 1992 to seek input and legislative ideas which would address Oklahoma rural health problems. The conference resulted in identifying twenty-six (26) health care issues, primarily personnel shortages, recruitment, and training issues, but also surfacing were preventive health, emergency care, ef-

Table 3. Rural Oklahoma's Health Care Concerns			
AHEC Regional 1991	**Governor's Conference 1992**	**OkRHA/Governor's Commission Regional 1993**	**OkRHA Annual Conference 1993**
Personnel	Personnel	Personnel	Personnel Define Mid-Level roles and advocate use
Licensing	Education/ Recruitment	Education/ Recruitment/ Licensing	Recruitment — via scholarships and bonuses Education — Fund rural training of primary care providers: NP's/PA's
Regulation Reimbursement No Insurance	Regulation/ Efficiency	Regulation/ Reimbursement	Efficiency — One Insurance Form
Home Health	Prevention	Prevention	Public Health and Prevention: Allow for health districts and enforce school health education
Emergency	Emergency	Medical Specialties	Emergency — Create statewide system
Transportation	Telemedicine	Telemedicine	Planning — State funding for local plans

ficiency, and telemedicine as important concerns. This conference awakened an even greater audience to the health care issues and concerns in the state of Oklahoma and was the impetus for the formation of the Oklahoma Rural Health Association.

Being a rural state, it is very important to seek input from the "grassroots" level to ensure that national and state legislation will address rural issues as well as urban issues. Because of the concern for grassroots input and to determine statewide health care priorities that include and overlap with local and regional concerns, the Oklahoma Rural Health Association (OkRHA) held four regional meetings in June of 1993. Nearly 200 participants actively provided input as to the health issues in rural Oklahoma and their priority level.

Community forums were held in each of the four quadrants of the state: in Claremore, McAlester, Lawton, and Enid with Woodward and Guymon participating via satellite hookup. Regional conference participants found it extremely difficult to rank issues, as many are serious in rural areas and are often interrelated. The top four rural health issues ranked in order of importance were:

- Shortage of health professionals and inadequate education, recruitment, and retention efforts;
- Restrictive regulations on hospitals and mid-level health practitioners, and inequities in reimbursement;
- Shortage of preventative and specialized health services; and
- Absence of health education, promotion and disease prevention activities.

These issues were taken and discussed at the Second Rural Health Conference, this time sponsored by the Oklahoma Rural Health Association (OkRHA) in October 1993. Using the input from previous forums, and current information and opinion on health care reform provided by state and national leaders at the conference, the following legislative and organizational issues were formulated by the participants and forwarded to the Board of OkRHA for action.

Legislative

1. Personnel
   a. Encourage recruitment and retention of health care providers by increasing incentives for choosing rural practice through signing bonuses, scholarships, tuition loan forgiveness and income tax credits;
   b. Increase funding and number of programs for training of primary health care providers, particularly advanced practice nurses, physician assistants and primary care physicians; and
   c. Tie funds for training to the number of graduates placed in rural communities.
2. Health Planning
   a. Provide adequate funding to develop statewide EMS/trauma system; and
   b. Provide funding for local strategic planning initiatives with requirements for full citizen participation, including the inclusion of minority and multicultural concerns.
3. Administrative and Regulative
   a. Require that health insurance companies, as a condition for licensing in Oklahoma, accept a single standard claim form used by all providers; and
   b. Allow for creation of health districts (financed through a county millage) for all health care facilities.
4. Prevention
   a. Enforce existing statutes calling for comprehensive health education in schools; and
   b. Provide funding for preventive health education for the elderly.

Organizational

1. Provide leadership in clarifying and defining appropriate scope-of-prac-

tice guidelines and reimbursement levels for mid-level health care providers;
2. Facilitate the development of a rural legislative caucus for rural health concerns;
3. Take the lead in defining "rural" for policy and legislative purposes; and
4. Develop active membership and education committees.

Table 4. Summary of Budget Study for EMS Alternatives for Binger, Oklahoma

I. Need for Service			80 Calls Per Year	
			Alternative 1	Alternative 2
II. Total Capital Costs			$44,470	$44,470
III. Annual Costs				
	Capital		$ 6,484	$ 6,484
	Operating		16,014	26,384
	Total		$22,498	$32,868
IV. Revenue				
	User Fees	Rate 1	$10,850	
		Rate 2	$16,450	
	District		$20,269	

Source: DeWitt and Doeksen

Future Directions

Although it has been illustrated that a viable health sector is important to the economies of rural communities, and the rural health problems as perceived by rural Oklahomans have been identified, the question remains, what can we do to help rural communities, providers, and facilities? Most of the problems and solutions suggested by rural providers and residents in Oklahoma imply changing external circumstances or receiving assistance from outside their communities. However, research by Amundson and his colleagues in the Pacific Northwest suggests a different type of solution is necessary, or that at the least a combination of solutions are required.

In one study of hospital utilization (the conclusions apply to the entire health sector), Hart, Rosenblatt, and Amundson (1989) conclude:

"...the two factors that play the most important role in [where to be hospitalized] are the location of the [patient's] personal physician and [the patient's] perception of the scope, relevance, and quality of services available in their local hospitals.... the presence of a local personal physician is associated with a much higher proportion of local hospitalizations. Those communities with the most severe physician shortages and with histories of physician instability are those where hospital market shares are the lowest."

And in a recent article, Amundson (1993) argues that it is the local community and providers that hold the keys to survival, not the state or federal government, or the state health professional schools. A community needs to be a desirable place to live in order to recruit health professionals, or any type of professional, or retiree. The hospital has to offer quality care, be an attractive enough place for someone to want to stay in, and have pleasant and helpful staff. The community has to have good schools, decent streets and sewer systems, and helpful merchants. Amundson would argue that we have met the enemy, and the enemy is (too often) us.

Specifically, rural hospitals can modify their scope of services and the way they deliver services and interact with the public. The emergency room is a place that most rural residents will use sooner or later, and the speed with which they are seen and the way they are treated when receiving care will influence their opinion of the entire hospital operation. The way bills are sent and collected has a substantial impact, even if only at the margin. Services can also be added or enhanced that will attract more of the younger local population. And clearly, public information that is disseminated about the hospital in the local newspaper influences public perception, particularly among that large segment of the population without recent personal interactions with the hospital or its staff. All of these factors affect both satisfaction and utilization, and all of them are to a large extent under the control of the hospital administration, staff, and board (Hart, et al., p. 14-16).

The recommendations by Amundson and Hart focus on mechanisms for reducing economic "leakages" to other communities, including urban areas. However, it is important to recognize that such a strategy will not always insulate the local health system from serious financial threats. Where such threats occur, there will be cases in which the community is no longer able to provide comprehensive health services. While this may be an undesirable outcome from the standpoint of the community's local economy, it may not necessarily have a negative impact medically—at least if the community residents have ready access to services in nearby communities. However, such communities do have at least two options other than closure; local subsidization and major restructuring of existing services.

While local subsidization through taxation is not that common, it is not unheard of. Indeed, the National Rural Health Association's 1992 Outstanding Rural Health Program Award was in recognition of the Ajo-Lukeville (Arizona) Health Service District (NRHA, 1993). There are also a number of examples in Oklahoma where counties and communities have voted a sales tax or property tax to support health services. This support has primarily been for health department services, hospitals, and EMS.

But even tax subsidy cannot ensure

that all vulnerable rural facilities will remain financially viable. Facilities supported by local tax subsidies have failed in Oklahoma. The second option open to the local community is a major reconfiguration of the local delivery system. Often, this reconfiguration involves the conscious decision to close or convert the local hospital or to enter into some form of multi-community collaboration, including hospital consolidation.

Examples of win-win situations are becoming more common, as illustrated by the following anecdotal observations from Colorado and Kansas:

"Closing a hospital can, in fact, lead to better overall health care services for a regional community. When poor financial conditions forced the closure of the Las Animas Hospital in Southeastern Colorado and, shortly thereafter, the neighboring Rocky Ford Hospital, community leaders faced a potential crisis. But the closures boosted the population base from which a third hospital in nearby LaJunta could draw, strengthening it. The changes also spurred the development of a comprehensive review of the regional health care needs. A national health care provider was attracted to the region and is now providing services in all three communities. Total health care employment in the area has actually increased since the two hospitals were closed (Sullivan, p. 1)."

And...

"The 2,500 citizens of [Caney, Kansas] recently lost the acute care hospital that... served families here for decades. The 24-bed facility had tried cost-containment, diversification into home services, and setting aside swingbeds for long-term care to shore up finances and maintain the traditional functions of a full service acute care hospital.

"But nothing worked to offset the stress from restricted reimbursement rates, a declining and aging population, and economically hard hit communities. In 1988, the board of trustees realized that the community could no longer support—nor did it need—a full service hospital. Instead of closing, the board revamped its delivery structure and today the people of Caney have access to primary care in a renovated facility. Residents can take advantage of new clinics offering cardiology, radiology, mammography, and laboratory services. Emergency services are readily available and the inpatient and outpatient surgery, and obstetric needs are met at a hospital 20 miles down the road.... Caney arranged with the Jane Phillips Hospital in Bartlesville, Oklahoma, to tend to the acute care needs of Caney's citizens. That shift gave Caney the chance to redirect its attention to primary care.

"Running the new clinic and its expanded primary care services cost $870,000 in 1990 and supported a staff of 33, compared with the $900,000 budget supporting a staff of 23 in the last year of the hospital's operation (Kimball, 1991, p.1)."

The common themes in the success stories include community involvement and strategic health planning. Many of us may not like the term planning and we can call it something else, but the bottom line is that we need community involvement in decision making. There are many excellent strategic health planning programs available. Professionals from the Office of Rural Health, Cooperative Extension Service, State Department of Health, AHECs, medical schools, and others are available to provide strategic planning programs.

An outstanding example is NWAHEC's adoption of PATCH in Noble County. By going through a strategic health assessment and planning process, the underlying community health problems were identified and innovative preventive programs implemented. The process involved the entire community, which meant all parties had input and all were kept informed and involved. That project continues even after NWAHEC has left the community.

In addition to helping with the strategic planning process, many agencies can offer technical assistance. In Oklahoma, the Cooperative Extension Service is heavily involved with budget evaluations of alternatives. For example, the rural community of Binger, Oklahoma, (population 724) was having difficulty maintaining EMS service. Their strategic planning process lead them through a process where they wanted to evaluate two possible service delivery alternatives. A summary of the analysis is provided in Table 4. Basically, the budget estimates the need for service. In this case, it is 80 calls per year. Second, the analysis estimates capital costs which includes an ambulance and radio equipment. Next, annual capital and operating costs are estimated, which yields a total of annual costs. Then the revenue sources are provided. The decision makers can now choose between alternative systems and revenue sources.

The point is that each agency has specific technical assistance available. Some have outstanding databases; others have funding information; still others have expert skills to share. Community leaders should not be shy in asking for assistance. The strategic planning process involves us all. We must take control of our own destinies. State and federal government must help by encouraging strategic health planning, passing enabling legislation and providing technical assistance; rural providers must serve as resource people on local committees; and local residents must take an active role in the planning process. It is essential when searching for creative solutions to local health care and economic development needs that the relationship between the provision of health services and the local economy be prominently recognized and considered. When this occurs, the end result can be an improved local economy, improved health services, and an empowered citizenry.

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The Authors

Gerald A. Doeksen, PhD, is a Regents Professor and Extension Economist at the Department of Agricultural Economics at Oklahoma State University (OSU). He specializes in rural development with emphasis on rural health issues.

Cheryl F. St. Clair is an extension associate with the Oklahoma Cooperative Extension Service in OSU's Agricultural Economics department. She is involved primarily with rural development infrastructure issues.

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Off the drawing board at last

PROklahoma to carry doctors' colors into Oklahoma managed care arena

IT'S OFFICIAL. The OSMA's managed care organization is being incorporated as PROklahoma (pronounced prokelahoma), a decision reached by the MCO's board of directors at its June 20 meeting in Oklahoma City.

The board selected the law firm of Andrews Davis Legg Bixler Milsten & Price, with offices in Oklahoma City, as counsel to organize and represent PROklahoma and an independent practice association to be named Oklahoma Physician Network (OPN).

C.L. Frates and Company, the Oklahoma City insurance agency that manages the Physicians Liability Insurance Company (PLICO) and PLICO Health, has been selected to provide initial management and administrative services for PROklahoma. The agreement stipulates that when the number of lives insured by PROklahoma reaches 5,000, the commitment to C.L. Frates and Company will be reassessed.

PROklahoma will be incorporated as a managed care organization that eventually will be able to offer a capitated HMOklahoma option, tailored managed care plans, or straight indemnity insurance, not unlike plans already available. What will set PROklahoma apart is utilization review and quality assurance *by physicians*. This, combined with 100% physician ownership, will make PROklahoma unique among Oklahoma carriers.

Participation in PROklahoma will be open to all qualified members of the Oklahoma State Medical Association and the Oklahoma Osteopathic Association. Details of membership fees for OPN and participation in PROklahoma will be announced in the weeks to come. Meanwhile, routine decision-making rests in the hands of PROklahoma's new executive committee: Chairman G. Lance Miller, MD, Tulsa oncologist; Eldon V. Gibson, MD, Shawnee internist; Jay A. Gregory, MD, Muskogee surgeon; David M. Selby, MD, Enid surgeon; and Michael L. Winzenread, MD, Oklahoma City family practitioner.

Speculation about a possible merger with Tulsa-based Physicians, Inc., also ended at the June 20 meeting. Physicians, Inc., Chairman Robert M. Melichar, MD, wrote to Dr. Miller, saying that his company does "not seek any dissolution or merging of Physicians, Inc., or Access Insurance with a separate OSMA entity." This effectively closed discussions with OSMA MCO representatives about forming a single, physician-owned health insurance company in Oklahoma. Melichar ended his letter saying, "We wish the OSMA MCO Committee, including ourselves as members of the OSMA, best of luck in its new endeavor."

The diverse objectives of the Tulsa-based organization and PROklahoma at this time do appear to preclude any joint venture: Physicians, Inc., is already licensed as an indemnity insurance company and as such must meet capital requirements established by the state insurance commissioner. PROklahoma, on the other hand, is being incorporated as an HMO and will seek certification through the Oklahoma State Department of Health. An HMO can offer a variety of health insurance products and is not obliged to meet the capital requirements of the state insurance commissioner.

At the AMA. Representing Oklahoma physicians, OSMA President Jay A. Gregory, MD, takes the floor during discussions at the June meeting of the AMA House of Delegates in Chicago.

Putting their heads together. At the AMA House of Delegates meeting in Chicago are Oklahoma delegates (from left) Edward N. Brandt, Jr., MD, Oklahoma City; Ed L. Calhoon, MD, Beaver; John R. Alexander, MD, Tulsa; and OSMA President Jay A. Gregory, MD, Muskogee.

It's your association

Bylaws not a "great read" but they dispel misinformation and clear the air

Like smoke from a fireworks display, questions lingered in the aftermath of the Annual Meeting of OSMA's House of Delegates last spring. A minor distraction, to be sure, but stinging the senses nonetheless.

Because some of the haze appeared to stem from a lack of knowledge of OSMA bylaws, and as a service to the many members who may be unfamiliar with them, the applicable chapter from the bylaws (revised in May 1993) is offered here in its entirety.

Chapter IV
House of Delegates

Section 1.00 Composition. The House of Delegates shall be composed of the general officers of the Association, Delegates and Alternate Delegates to the American Medical Association, Trustees and Alternate Trustees, Delegates elected by the component societies, Delegates elected by the sections recognized in Section 1.04 below, all Past-Presidents of the Association, and the Deans of the recognized medical schools in Oklahoma, provided they are members of the Association.

1.01 Qualifications. Delegates and Alternate Delegates must be members in good standing of the Oklahoma State Medical Association.

1.02 Representation. One Delegate and one Alternate Delegate shall be elected by component societies for each 25 Active, and Active Limited Dues Members residing in the State of Oklahoma, or fraction thereof.

1.03 Determination of Representation. The number of Delegates from a component society shall be determined by the records of the Association on December 31 of the year preceding the term of service.

1.04 Section Representation. The following sections shall be entitled to one Delegate and one Alternate Delegate each: Young Physician Section, Medical Student Section, Resident Physician Section, and Hospital Medical Staff Section.

Section 2.00 Election and Term of Office. Delegates and Alternate Delegates shall be elected in November or December by component societies and special sections. A List of Delegates and Alternate Delegates shall be certified to the Association on or by January 15 following their election and shall continue in effect for a period of one year. The component society shall certify any Past-Presidents residing or practicing in their area as additions to their Delegates.

Section 3.00 Meetings, Attendance and Duties.

3.01 Annual Meeting. The House of Delegates shall conduct its annual meeting at the time and place selected by the Board of Trustees in compliance with Chapter III, Section 1.00.

3.02 Special Meetings. Special meetings may be called by the President, or by the Board of Trustees, or by petition to the Speaker from not less than 30 Delegates. Only such business as is specified in the call shall be transacted at a special meeting.

3.03 Quorum. A majority of the certified and qualified Delegates shall constitute a quorum at any annual or special meetings, provided that the Past-Presidents shall not be included in the quorum list for Delegates. (Editorial Note: The purpose of this last provision is to allow the Past-Presidents that are present to be counted as a part of the quorum, but not to include the Past-Presidents in the official list so as to change the quorum number.)

3.04 Attendance. Any member of the Association and invited guests may attend a meeting of the House of Delegates.

3.05 Separated Sessions. When justified by the volume of business to be considered at an annual meeting, the Speaker may hold the opening and closing sessions of the House of Delegates on separate days.

3.06 Duties. The House of Delegates shall conduct the necessary elections of officers and members of the Board of Trustees and shall transact all business as properly brought before it and other business as given by these bylaws or as brought by the Board of Trustees.

3.07 Privileges. Only credentialed Delegates or their seated alternates, or chairs of Association councils and committees, acting in their official capacity, may introduce business into the House. The Speaker of the House of Delegates shall determine who has the privilege of the floor.

3.08 Elections. All elections conducted by the House of Delegates shall be by majority vote. A nominee that has received a vote on a majority of the legal ballots cast shall be declared elected.

Section 4.00 Committees.

4.01 Reference Committees. Prior to the annual session, the Speaker shall appoint Reference Committees from the membership of the House of Delegates, which committees shall act to expedite the business of the annual session by recommending to the House what action should be taken on business referred to it. At special meetings, the option of appointing reference committees shall rest with the Speaker.

4.02 Credentials Committee. The Speaker of the House of Delegates shall annually appoint a Credentials Committee, whose members must be Delegates.

4.021 Credentials. The Chief Executive Officer shall certify to the Credentials Committee a list of Delegates, Alternate Delegates, general officers, Past-Presidents, and AMA Delegates and Alternate Delegates.

4.022 Duties. The committee shall determine the eligibility of the certified Delegates as representative of their component societies, certify such Delegates and determine the presence of a

Dr. Mary Anne McCaffree (second from left), speaker of the OSMA House of Delegates, takes her place on the dais at the AMA's annual meeting in June.

quorum prior to the House of Delegates proceeding with the transaction of its business.

4.023 Contests. In the event of a contest between alleged Delegates from a component society, the committee shall hear all relevant facts, adjudge who shall be Delegates, and report its findings and judgment to the House of Delegates, which may be accepted, rejected or otherwise disposed of by the House of Delegates. Contested Delegates shall not be seated until their status is resolved.

SECTION 5.00 Election of Delegates to the American Medical Association. The House of Delegates shall elect Delegates and Alternate Delegates to the House of Delegates of the American Medical Association in accordance with the Constitution and Bylaws of that body. They shall be elected at the annual meeting for a term of two years commencing at the close of the annual meeting at which elected. Such Delegates or Alternate Delegates shall attend all meetings of the American Medical Association House of Delegates.

5.01 Nominations. Any physician-member of the OSMA wishing to run for a position of Delegate to the AMA must have served at least one term as an Alternate Delegate or have served as an officer of the Association. There shall be at least twice as many nominations as there are AMA Alternate Delegate offices. Any physician member of the OSMA wishing to run for a position of Delegate or Alternate Delegate must be an active member of the Association as specified in Chapter I, Section 2.01, and provide a letter of nomination signed by a member of the OSMA House of Delegates to the Association office in Oklahoma City not less than 30 days prior to the election. If 25 days prior to the election, there are not twice as many candidates as (Alternate Delegate) seats or positions up for election, then the Executive Committee of the Association shall solicit candidates who desire to run and hold office in order to obtain twice as many candidates as there are seats available.

5.02 Elections. All candidates for AMA Delegate and Alternate Delegate shall run at-large and each member of the House of Delegates shall vote for one candidate for each of the Delegate and Alternate Delegate positions up for election. (Example: If there are four Delegate and four Alternate Delegate positions open, each voting member of the House will cast four votes for four candidates for Delegate and four votes for four candidates for Alternate Delegate.) As soon as any candidate obtains a majority of those voting, he/she shall be declared elected. Balloting will continue until all of the Delegate and Alternate Delegate positions are filled by individuals obtaining at least a majority. Approximately one-half of the Delegate and Alternate Delegate positions shall be selected each year. In the event a new Delegate and Alternate Delegate position is created, its first term shall be either for one or two years, whichever is appropriate to allow the House to continue to choose approximately one-half of the Delegate and Alternate Delegate positions each year.

5.03 Vacancy. If any office of Delegate is vacated, the Board of Trustees of the Association shall choose an Alternate Delegate to succeed to the position for the remainder of the unexpired term. If there is not time for the Board of Trustees to choose an Alternate Delegate before a meeting of the AMA House of Delegates, an Alternate Delegate shall be chosen to serve as Delegate for that session by the President of the Association. If an office of Alternate Delegate is vacated, the President of the Association shall appoint an OSMA member to serve until the next annual meeting, at which time a successor shall be elected to fill the unexpired Alternate Delegate term.

SECTION 6.00 Component Societies.

6.01 Charter. The House of Delegates may authorize the issuance of charters to county societies or district societies which shall be component societies of the Association.

6.02 Revocation. The House of Delegates may revoke the charter of a component society for cause.

SECTION 7.00 Trustee Districts. The House of Delegates shall divide the state into suitable trustee districts, based upon a two-thirds vote of the Delegates present.

SECTION 8.00 Finance.

8.01 Budget. The House of Delegates shall consider and act upon the budget submitted by the Board of Trustees.

8.02 Dues. The House of Delegates shall assess Association dues.

8.03 Assessments. The House of Delegates shall have the power to levy special assessments, and to stipulate the time and method of payment.

SECTION 9.00 Order of Business. The following shall be the general order of business at all sessions of the House of Delegates: (1) Call to order by the Speaker; (2) Invocation; (3) Report of the Committee on Credentials; (4) Presentation, correction and adoption of the minutes; (5) Report of officers; (6) Report of committees; (7) Unfinished business; (8) New business. The order of business of the annual meeting of the House of Delegates shall be set by the House on recommendation of the Speaker.

SECTION 10.00 Memorials and Resolutions.

10.01 Initiation. A memorial or resolution may be initiated by a component society or by a member of the Association, and must be signed by either the secretary of the component society or by the member, respectively.

10.02 Filing. All memorials and resolutions to be considered by the House of Delegates must be filed with the Executive Office of the Association at least 30 days prior to the meeting at which they are to be considered. The Board of Trustees may initiate, or may recommend to the House of Delegates a memorial or resolution from a component society, or by a member of the Association, waiving the requirement for prior filing.

10.03 Approval. All memorials and resolutions approved by the House of Delegates shall be in the name of the Association.

10.04 Delegated Authority. When the House of Delegates is not in session, resolutions may be issued by the Board of Trustees in the name of the Association and such actions shall be reported to the House of Delegates at its next meeting.

10.05 Certificates of Accomplishment. Accomplishments in the field of medicine may be recognized by a Certificate of Accomplishment. Such award shall be initiated by a component society, approved by the Board of Trustees

Bylaws *(continued)*

prior to the annual meeting, and voted by the House of Delegates.

SECTION 11.00 Special Sections. The special sections, as recognized in these bylaws, shall be organized and shall function according to rules established by the Board of Trustees. The rules adopted shall include, but shall not be limited to, a mechanism allowing each section to choose its Delegate and Alternate Delegate. Provided, however, that the Trustees may impose different mechanisms on different sections as needed.

LETTERS

Does AMA represent Oklahoma physicians?

TO THE EDITOR: I presented the resolution to deunify last year for several reasons, not the least of which was to make the AMA more representative, hence a better organization. The AMA has proven to be no different than any lobbying group, that is, looting federal coffers for the membership through compromise and lacking principle. The physicians of Oklahoma would be much better served by the AMA if it took a principled stand, not a utilitarian, expedient, compromising one. The stand on homosexuality by the AMA so objectionable to Dr. Lester and others is an example of the AMA taking a stand without principle simply to prevent alienation of the socialists in the White House and those contacts in the Congress that have been supportive of organized medicine's looting of the federal coffers. The same goes for the original stand on employer mandated health coverage. Any beginning student of economics understands the relationship between requirements put on small businessmen and unemployment. I suggested in a letter to Dr. Todd that the AMA simply announce its support for unemployment, as this is the natural and inevitable result of mandating employer-provided health insurance coverage.

Probably the most devastating effect that a lack of principle has had on the practice of medicine is the failure of the AMA to argue the immorality of price controls in the practice of medicine. Rather, the *level* at which the prices were set and controlled was the tack that the AMA took in a compromise. I can just hear the AMA bureaucrat: "Okay, we will go for price controls but a committee must be set up with physician participation to set those prices." What a disaster for physicians and patients, particularly the elderly patient.

The AMA gained entrance to the debate on health care during this administration only due to the efforts of the American Association of Physicians and Surgeons. Their suit against Mrs. Clinton and her closed door hearings policy allowed the AMA to send its representative to these meetings. The AMA has surely lost its position as a source of information about how its membership thinks and has flourished as a typical PAC institution, willing to compromise for a partial win even though basic principles are discarded. I noticed that the president-elect of the AMA, one Lonnie Bristow, declared that his charge as president will be to "implement whatever health care reform passes in the Congress." Now that's taking a stand for the membership.

I would argue that the AMA would be more representative if its membership were not cut and dried. If the AMA had to prove its ability and willingness to represent Oklahoma physicians, the organization would be better and would better serve us in Washington. The leadership of the OSMA and AMA have made it difficult for the average physician to become an AMA delegate and assume a position to change the current situation. One must serve as an officer in the OSMA

Medical Practice Act revised

Practice Act allows higher copy charges

RECENT REVISIONS to Oklahoma's Medical Practice Act include an increase in the amount physicians may charge patients for making copies of their medical records.

The new law, effective September 1, 1994, sets the maximum charge at 25 cents per page. Previously, physicians were allowed to charge no more than 10 cents per page.

The relevant portion of the act reads as follows:

Any person who is or has been a patient of a doctor, hospital or other medical institution shall be entitled to obtain access to the information contained in all his medical records upon request, and shall be furnished copies of all records pertaining to his or her case upon request and upon the tender of the expense of such copy or copies. Cost of each copy shall not exceed twenty-five cents ($0.25) per page. The physician, hospital or other medical professionals and institutions may not charge a fee for searching, retrieving, reviewing and preparing medical records of the person in order to determine which medical records are to be copied. Provided that this entitlement to medical records shall not apply to psychological or psychiatric records. In the case of psychological or psychiatric records, the patient shall not be entitled to copies unless access to said records is consented to by the treating physician or practitioner or is ordered by a court of competent jurisdiction upon a finding that it is in the best interest of the patient... or if the patient is a minor child or a guardian has been appointed for the patient, the guardian of the patient may authorize the release of the psychiatric or psychological records of the patient to a third party payor or governmental entity. The execution of such authorization shall not be construed to authorize the patient personal access to said records or information.

HEALTH DEPARTMENT

OSDH issues rules for reporting lead poisoning in children

EVIDENCE of adverse effects of lead at concentrations previously thought safe prompted the Centers for Disease Control and Prevention (CDC), in October 1991, to lower the acceptable level for blood lead levels in children under age 6 years to 10 µ g/dL. Both the American Academy of Pediatrics and the CDC recommend risk assessment of all children 6 months to 72 months of age at each health supervision visit, and universal screening of all children at 1 year of age and, if possible, at 2 years of age. Screening based on risk status is acceptable only in geographic areas designated by state health departments where adequate data for universal screening have established that there is a very low prevalence. At this time, no area in Oklahoma has been designated as very low risk. In response to these new recommendations, the Medicaid EPSDT Program now requires risk assessment and initial blood lead testing of all children ages 6 months to 72 months of age and subsequent blood lead testing based on age and risk in accordance with the CDC guidelines.

While childhood lead poisoning screening can be accomplished by collecting either a capillary or venous blood lead specimen, the ease of collection of the capillary specimen should enhance compliance with the new recommendations. Screening for lead poisoning is feasible by direct measurement of lead concentration in blood samples obtained by fingerstick technique after careful attention to cleaning the skin.

Comparisons of fingerstick blood lead levels and venous blood lead levels have demonstrated that the fingerstick screening test is accurate at lower levels and the false positive rates obtained are acceptable.

In July 1993, the Oklahoma State Department of Health modified its reporting requirement for lead poisoning by lowering the level for reporting blood lead levels in children and adults to 10 µg/dL. All health care providers and laboratories are now required to report individuals with levels at or above 10 µ g/dL. The Reportable Disease Card (Rev. 2/92) from the Oklahoma State Department of Health should be used by health care providers for reporting.

For additional information on childhood lead poisoning prevention or reporting, contact the Pediatric Division, Child Health and Guidance Service, Oklahoma State Department of Health, 1000 Northeast 10th Street, Oklahoma City, OK 73117-1299 or call toll-free statewide 1-800-766-2223, or in metro Oklahoma City, 271-4471.

or be an OSMA delegate for a certain length of time in order to be eligible for an AMA delegate position. This is an elitist policy that leaves Oklahoma physicians no alternative but to relinquish our unified status with AMA. If the vast majority of physicians in Oklahoma support the AMA and willingly belong, then mandatory membership is superfluous. The AMA deserves only those members who are not coerced to join. That so many physicians are willing to jump the hurdle of mandatory AMA membership to belong to their state/county society speaks well of the membership. The AMA should no longer be allowed to hide behind the carrot (PLICO Health) dangled in front of the membership.

—G. Keith Smith, MD
Oklahoma City

[Note: OSMA bylaws do not require a member to have been an officer or delegate in order to become an *alternate* delegate to the AMA. The applicable chapter of the bylaws begins on page 376.

Part of the JOURNAL's mission, as stated annually in its report to the OSMA Board of Trustees and House of Delegates, is "to serve as an open forum for the expoloration and discussion of issues vital to the physicians of Oklahoma." Readers are encouraged to express their opinions in this column. Letters should be addressed To the Editor, JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION, 601 Northwest Expressway, Oklahoma City, OK 73118. *—Ed.]*

CAPSULES

■ Health insurance does *not* replace workers' compensation insurance in the state of Oklahoma, notes the June *PLICO News*. Physicians' employees, by law, must be covered by workers' compensation insurance. An OSMA-endorsed workers' comp program is available through C.L. Frates and Company in Oklahoma City.

■ Perry A. Lambird, MD, Oklahoma City, an announced candidate for vice-speaker of the AMA House of Delegates in 1995, has so far drawn one opponent for the position—Dr. Allen S. Edmonson of Memphis, Tenn. It is anticipated that additional candidates may be announced from the states of Texas, Georgia, Ohio, and Pennsylvania.

Deaths

Robert Bruce Howard, MD
1913 - 1994

OSMA Life Member Robert B. Howard, MD, died June 16. 1994, in Oklahoma City. Dr. Howard, a native of Oklahoma City, was graduated from the University of Oklahoma School of Medicine in 1936. He became a general surgeon and practiced in Oklahoma City until his retirement in 1987. During World War II Dr. Howard served on active duty with the U.S. Army overseas. In addition to his active practice, he was a member of the Isotope Committee and assistant professor of surgery at his alma mater.

George Loren Norris, MD
1909 - 1994

George L. Norris, MD, retired Bartlesville internist and cardiologist, died March 27, 1994. Dr. Norris was born in Olathe, Kans., and attended the University of Kansas School of Medicine, where he was graduated in 1940. He completed an internship in Albany, N.Y., and a residency at the University of Washington School of Medicine in St. Louis, where he was a fellow in clinical cardiology. Dr. Norris practiced medicine in Winfield, Kans., from 1941 to 1961, a period that was interrupted by two years of active duty with the U.S. Army Air Force during World War II. He moved to Bartlesville in 1961.

Harold Ray Sanders, MD
1913 - 1994

Longtime Stillwater obstetrician-gynecologist Harold R. Sanders, MD, died June 15, 1994. The Rocky, Okla., native was a 1943 graduate of the University of Oklahoma School of Medicine. After two years in the military he settled in Konawa to practice and in 1947 moved to Stillwater. In 1965-66 he served as chief of ob/gyn for the government of American Somoa in Pago Pago and taught family planning to the islanders. Dr. Sanders retired in 1979 and was awarded Life Membership in the OSMA.

Wesley A. Whittlesey, MD
1936 - 1994

Arlington, Va., native Wesley Whittlesey, MD, died July 12, 1994, at his home in Oklahoma City. Dr. Whittlesey was a 1963 graduate of the University of Oklahoma College of Medicine. After completing his postgraduate training in pediatrics at Stanford Medical Center in California in 1966, he returned to Oklahoma to accept a post as director of pediatric service at the state health department. Later he was assistant supervisor of medical units at the Department of Human Services, inpatient coordinator at Oklahoma Children's Hospital, and medical director of Blue Lincs HMO. Dr. Whittlesey also was a clinical associate professor of pediatrics at the University of Oklahoma.

In Memoriam

1993

John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Edward David Greenberger, MD	August 6
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13
Earl Mathiews Woodson, MD	February 20
Tom Lamar Johnson, MD	March 5
Orville Main Rippy, MD	March 11
Minor Elliott Gordon, MD	March 14
George Loren Norris, MD	March 27
Max A. Glaze, Md	April 29
Winfred Aaron Showman, MD	May 14
Carter William Mathews, MD	June 3
Harold Ray Sanders, MD	June 15
Robert Bruce Howard, MD	June 16
Wesley A. Whittlesey, MD	July 12

Classifieds

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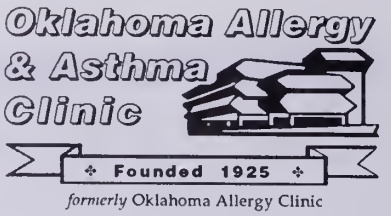

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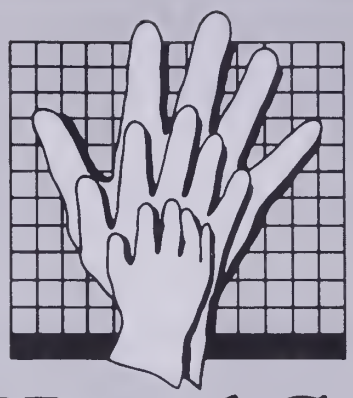

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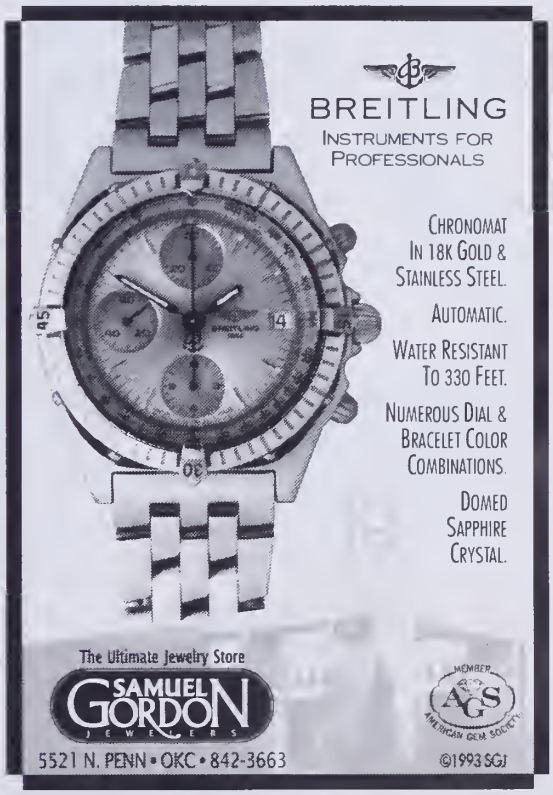

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The Last Word

■ **First Lady Hillary Clinton met with a group of Okla**homans June 24 in Wash-ington, and among them were seven members of the Oklahoma State Medical Association (OSMA). William L. Corporan, MD, Duncan; Roy L. Dehart, MD, John I. Fishburne, MD, and Edd Rhoades, Jr., MD, Oklahoma City; F. Daniel Duffy, MD, and John B. Nettles, MD, Tulsa; and Paul B. Edmonds, MD, and Don L. Wilber, MD, Midwest City, met with Mrs. Clinton in the Executive Office Building for 45 minutes, following a two-hour briefing on the Clinton plan for health system reform. Ira Magaziner also addressed the group. The physicians stressed the need for meaningful tort reform and expressed concern for the future funding of medical education and research, while Mrs. Clinton sought the support of Oklahoma Senator David Boren and Representative Bill Brewster for the President's plan. The meeting was one of a number of White House visits with delegates from states whose legislators serve on either the Senate Finance Committee or House Ways and Means Committee.

■ **Muskogee family practitioner Tom Coburn, MD, has** announced his candidacy for Oklahoma's Second Congressional District seat. The race will pit the Republican Coburn against Democratic incumbent Mike Synar, a formidable opponent in a district that hasn't elected a Republican since 1920. A political conservative, Coburn has said he thinks health system reform should focus on insurance reforms, medical savings accounts, and subsidies for the needy. Coburn is a 1983 graduate of the University of Oklahoma College of Medicine.

■ **Saturday, September 24, the Cimarron National Golf** Club in Guthrie will host the first Feed the Children Golf Classic, and Oklahoma physicians are invited to participate. Celebrities from the worlds of sports and entertainment will join physicians and other players in a golf scramble beginning at noon. Registration is $165 per person and is tax deductible. For entry forms or information, call Cynthia Judd or Karen Moore, (405) 942-0228, or write Feed the Children, P.O. Box 36, Oklahoma City, OK 73101. Cosponsors of the event are the Oklahoma State Medical Association and Manners, Goosby & Olson.

■ **A new medical publication has been launched by Rich**ard L. Reece, MD, Oklahoma City pathologist, and David H. Combes, president of Deaconess Clinical Associates. *The Oklahoma Medical Voice*, a weekly newspaper dedicated to coverage of the health care profession at both state and national levels, will be distributed free of charge throughout the Oklahoma City area. Editor and publisher of the newspaper will be Brian Sherman, longtime editor of *Private Practice*, a national medical magazine that recently discontinued its Oklahoma City-based operation. Reece, a relative newcomer to Oklahoma, is the former editor-in-chief of *Minnesota Medicine*. After moving to Oklahoma several years ago, he continued to publish his own monthly newsletter, *The Reece Report*. His tongue-in-cheek style became familiar to JOURNAL readers last year in "A Letter to the Clintons About Lawyers and the Principles of Managed Competition" (June) and "Open Letter to President and Hillary Rodham Clinton—This Time About Total Quality Management for Lawyers and About Running Courthouses Like Hospitals" (September).

The Oklahoma Medical Voice office is on the tenth floor of the JRC Medical Tower, 3141 Northwest Expressway, Oklahoma City, OK 73112.

■ **OSMA members who have not yet submitted the ques**tionnaire on OSEEGIB reimbursement changes are encouraged to do so. The forms, published in the June *OSMA News*, ask you to list your ten most often used CPT codes, your normal charge, and both your 1993 and 1994 OSEEGIB reimbursements. The OSMA Council on Member Services needs this information in order to deal with OSEEGIB and the legislative committee that will investigate OSEEGIB this fall. Please note that you are being asked to provide your OSEEGIB reimbursement figures to the OSMA. Some forms have been returned without the OSEEGIB figures, indicating that perhaps the respondents thought the OSMA was to supply the figures.

■ **The controversial Supervised Medical Doctor (SMD)** designation required by the state's medical practice act was unceremoniously laid to rest earlier this summer. Meeting in June, the Oklahoma State Board of Medical Licensure and Supervision implemented the Legislature's revised medical practice act on an emergency basis pending review by the Governor, Legislature, and public. The revised act was signed into law during the Legislature's recent session.

■ **The 1994 Edward R. Munnell Award for outstanding** service in smoking and health programs was recently presented to OSMA Associate Director Mike Sulzycki by the American Lung Association of Oklahoma. Sulzycki, who chairs the Tobacco Free Oklahoma Coalition, received the award at the ALA's annual meeting in Oklahoma City June 25. The award is named in honor of OSMA Life Member Ed Munnell, MD, longtime Oklahoma City thoracic and cardiovascular surgeon.

■ **Any physician serving on the board of directors of an** HMO or PPO needs to report that information to PLICO. Currently there is an exclusion in PLICO's professional liability coverage for such physicians, but a special policy is being made available to provide the coverage. Interested physicians should call Del Langham at PLICO, (405) 524-0801.

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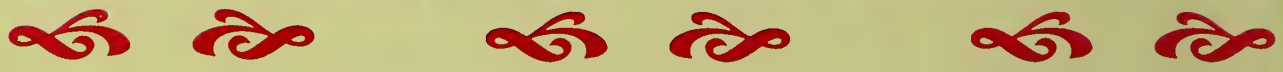

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The JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125.

POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscriptions to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single copies are $3 per copy, prepaid, subject to availability.

Reprints of articles are available either from the authors or from University Microfilms International, 300 North Zeeb Road, Dept. P.R., Ann Arbor, MI 48106, 1-800-521-3044.

National advertising representative: State Medical Journal Advertising Bureau, Inc., 11437 West 106th Street, Overland Park, KS 66214, (913) 888-8781, 1-800-788-2360.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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ABOUT THE COVER

Foliage in Red Rock Canyon reflects September's change of season.

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physical move later this year

Tricycle or Bicycle?

A rancorous spat about unified membership at the Annual Meeting is a reminder that mandatory participation is an issue that is never quite resolved. For as long as anyone alive can remember, there have been periodic and recurrent ructions on the subject, with variable amounts of thunder and lightning, but usually with a vote of one-fourth to one-third favoring deunification and a fairly solid majority always favoring continued unification. The heat of the debate and the recurrent nature of the battle justifies a moment of introspection on the etiology of such a persistent question.

A medical association is not a labor union, but often is required to function like a union in the marketplace. While the labor union can successfully insist that its members pay dues or not work, the medical association is disconnected from the medical licensing process, and must carry on by offering benefits other than the opportunity to make a living.

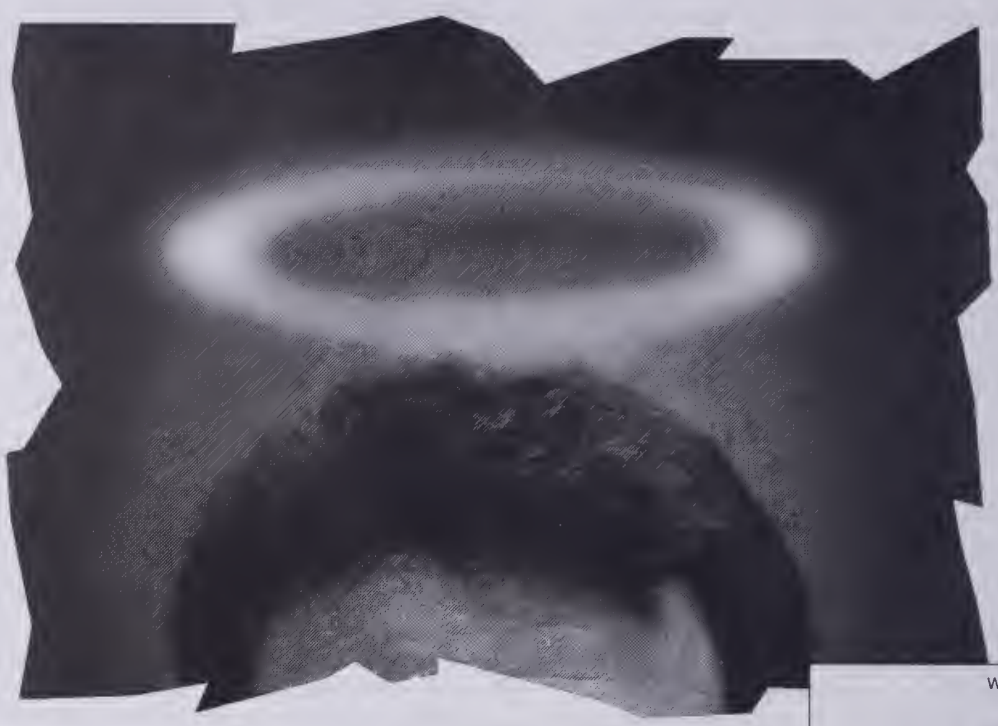
Also, the three components of the medical association differ in form and in many functions from the labor union. The county medical society sometimes seems to be little more than a "meet and eat" club, but the collegiality formed there is the foundation block for all of medicine's more complex actions. The state association is the plane of effective interface of most physicians with the officialdom of the community and local governments. On the other hand, Congress and the President's Administration are not influenced by state medical associations but will sometimes listen to the American Medical Association on national medical policies and long-range planning.

It is evident that a county society cannot influence Congressional deliberations, and it is also evident that the AMA cannot command the collegial function of the county society , nor affect the state association's effort to lobby a legislative bill. Thus we can see that the three levels of medical association activity are functionally so different that none can substitute for another. Each part is essential, yet none can stand alone. Like the three wheels of a child's tricycle, each component of the trinity medical association contributes uniquely to the total function. If we take away any wheel of the tricycle, we do not have a functional bicycle, but an inoperative piece of junk. Physicians share several very human characteristics of mankind that transcend the medical organization. Any particular physician's **need** of membership in a medical society will vary from time to time. Any physician's **use** of a medical society will vary from time to time. Every physician's **knowledge** of medical society activity will vary from time to time. Any physician's ability **to appreciate** the medical society is dependent on the physician's understanding of the current issues being addressed. At any given moment in time there are some of us with little use for the labor union function of the association, and negative votes then result.

It is unfortunate that medical society membership costs money; unification friction would probably disappear if there were no dues. But if we are to "meet and eat" we must buy the food, and likewise we must provide the funds for the more complex activities. So long as the association must function at times as a physician's labor union, the physician will have to pay "union dues." The choice the physician has is to **not** associate, or to associate with an ineffective labor union, or to associate with an effective labor union. Fortunately, the Oklahoma State Medical Association remains a democratic organization, and a periodic vote on unification versus deunification will continue to keep the association's functions congruent with the members' collective desires. Let's stop snarling at each other about unification and get on with the constructive work of the association.

Ray V. McIntyre, M.D.

Physicians who refer to us acquire a certain reputation with their patients.



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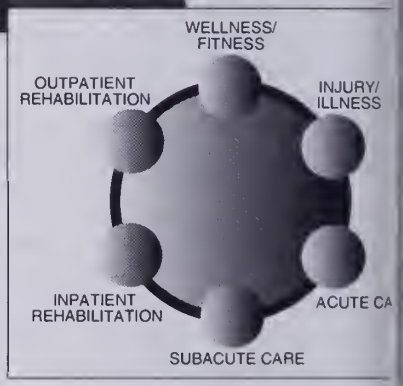

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Eh, What's Up, Doc?

Well, by the time you read this page, I am confident that a lot of rhetoric (the Okie word is *B.S.*) concerning health system reform, the elections, and the darned AMA, etc.... will have gone by the wayside. Enough of my lectures from the pulpit for now (I didn't have any good inspirations. Let me bring you up to date on three areas of significant interest to all physicians.

First, the AMA convened a consortium, "The Study of the Federation," the purpose of which is to initiate the process of envisioning a federation—or configuration of relationships, processes, and structures—that would be responsive to the future needs of physicians and health care professionals. The first meeting of the consortium was May 26 in Chicago. I am pleased to inform you that ours was one of six states with whose representatives the consultants held an in-depth meeting for input into the study. That meeting was August 19, and I would like to thank Drs. Deborah L. Huff, John L. Aldridge, M. DeWayne Andrews, Philip Mosca, Ray V. McIntyre, and Gary L. Paddack for their participation in the state focus study group. Other meetings will be held with national specialty societies, large county medical societies, and other medical leaders and staff to develop a broad data base. A very high degree of support was generated at the May meeting by the 162 participants as to what characteristics the "future house of medicine" should possess. Some, but certainly not all, of those characteristics were:

Credibility and reliability in the eyes of the public
Responsiveness to the diversity among physicians
Organization which generates trust
Stable financial base
Strategic alliances; coalitions
Pro-active organization, with well-defined goals

I will attempt to keep you abreast of this most interesting project as it unfolds.

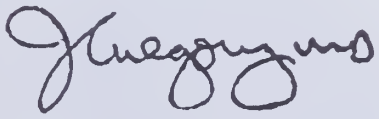
Next, let me discuss the unholy trinity of the AARP/AFL-CIO/AMA. Many of you, with good reason, have discussed this issue with me and some directly with the AMA leadership. Needless to say, the formation of the above coalition was undertaken without enough consideration being given to the full ramifications such a coalition would have with members of Congress, as well as with members of the AMA. I can say that this act appears to be the work of a policy lawyer who works on staff at the AMA. I can also say that the AMA leadership would probably not form this coalition if they had it to do over again. The good that came from the coalition was that the AARP and the AFL-CIO came out in support of the AMA's Patient Protection Act. But in doing so, the press release gave the impression that we, the AMA, were backing employer mandates and a comprehensive benefits package for all. As you know, we have softened greatly our stance on employer mandates, and we speak in terms of basic benefits packages, not comprehensive packages. Our concerns have been addresses by the leadership and we must continue to keep the pressure on the AMA so that such a faux pas does not occur in the future. I would like to thank my classmate Jeep Dewberry, MD, Oklahoma City, for his vigilance on this issue.

Let me say a few words about the MCO/IPA. As of this writing (Aug. 19), bylaws for the MCO/IPA are being distributed to their respective boards for comment and adoption. License applications with the appropriate regulatory bodies are being made. And the explanatory video, which all of you have or soon will receive, is in production. If it seems that progress has been slow over the past two years, you are correct. But, my friends, this is probably our last chance to create an entity which only you and I will own, an entity in which you and I will assume ALL control, and an entity which serves ALL of us equally and in which no one will benefit unduly from the work of another. This project will be successful, but it is going take all of our collective effort to pull it off.

I am sympathetic to and understand the "conflict of interest" with other managed care plans that many of our colleagues have cited as their reason for not helping in the formation of this entity. You must understand that this is the only entity that will protect all of us in the future. Your PHOs and PHO hybrids, or hospital-based PPOs, etc.... give much of the control to the hospitals, administrators, or other entrepreneurs. The for-profit ventures will benefit only those who have the money to invest heavily. Such ventures in many other states have sold out to big business if and when they became profitable, leaving the network of minimally invested physicians to provide the health care. Our MCO/IPA is for all of us, not benefiting any one of us more than another. You must understand this very key, fundamental element; it is this element that makes our project different from any other. PLICO was founded on the same principle, and PLICO continues to be successful, with all decisions being made by a board that is elected each year by you, the shareholders of the company.

Some have said that primary care is crucial to the MCO/IPA, and that is true; no more so, however, than the day-to-day care of our patients. Specialists need a solid core of primary care physicians, just as primary care physicians need a core of specialists, to provide specific services if and when the need arises. We must all work together, for when we do, we are a formidable force indeed. Remember, throughout this endeavor, we must be PROklahoma!

Have a great day!



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Androgenic/Anabolic Steroid-Induced Intrahepatic Cholestasis: A Review with Four Additional Case Reports

Ahmet Gurakar, MD; Paolo Caraceni, MD; Stefano Fagiuoli, MD; David H. Van Thiel, MD

Four cases of severe cholestasis attributed to anabolic/androgenic steroid usage are reported. These cases are reported because each was severely jaundiced (peak bilirubin level 62 mg/dl), developed advanced hepatic failure (Child's Class B) and was referred to a liver transplant center for consideration for liver transplantation. The hepatic dysfunction and cholestasis in each persisted for 3 months or more. Moreover, in two the hepatic dysfunction was complicated by the development of renal dysfunction and anemia requiring additional medical interventions. With prolonged medical therapy, each case recovered fully without transplantation. These cases are important because they demonstrate that drug-induced cholestasis can be prolonged, can mimic advanced liver disease, and can be associated with co-existent renal dysfunction.

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Oklahoma 73112.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112-4481.

ANABOLIC steroid-induced hepatocellular cholestasis is a well-known entity. It was described originally in 1947 by Werner.[1] Although initially it was thought to be an idiosyncratic hypersensitivity reaction,[2] it is now recognized as an intrinsic direct hepatotoxicity of anabolic steroids that is dependent on individual susceptibility, particularly genetic factors.[1,3]

Although a case of intrahepatic cholestasis produced by a nonalkylated anabolic steroid (19-norandrostenolone) has been reported to have occurred in a 27-year-old pregnant woman during the third trimester of her pregnancy,[5] C-17 alkylated anabolic steroids have been implicated in the pathogenesis of the majority of cases.[4]

The present communication reports four additional cases of androgenic/anabolic steroid-induced cholestasis seen by the authors between 1985 and 1991. Each case was referred to the authors for possible liver transplantation because of perceived hepatic failure occurring in an individual as a consequence of prolonged severe cholestasis.

Case Reports

Case 1.—A 63-year-old male with a past history of a left lower lobe lobectomy for bronchiectasis at age 35 and a recent complaint of impotence was treated with methyltestosterone. Six weeks after the initiation of this therapy, he developed a progressive cholestasis that continued despite his having discontinued the drug. He was admitted to the hospital with symptoms of progressive lethargy, jaundice, pruritus, diarrhea, and a 35-lb. weight loss, all of which had occurred

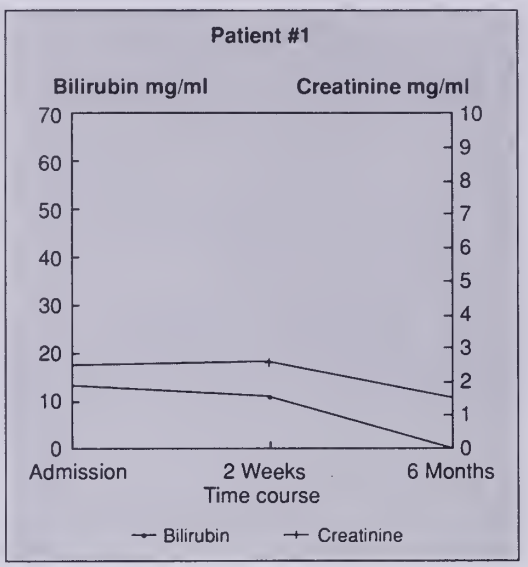

Figure 1. Serum bilirubin and creatinine levels in Case #1 shown at admission, 2 weeks and 6 months during the course of their illness.

When used in therapeutic doses, anabolic steroid produces clinical jaundice in only a small number of recipients.

since the initiation of his anabolic steroid treatment. He denied any nausea or vomiting. He admitted to social alcohol use but denied any history of smoking, intravenous drug abuse, sexual promiscuity, or prior blood transfusions. Physical examination revealed an emaciated white male with hepatomegaly, jaundice, widespread skin excoriations, and a mild encephalopathy.

His initial biochemical evaluation revealed a total bilirubin level (TB) of 13.2 mg/dl (normal value 0.2-1.3) with a direct fraction of 9.8; alkaline phosphatase (AP) of 337 IU/L (normal value <290); gammaglutamyl transpeptidase (γ GGT) of 53 IU/L (normal value <44); SGOT (AST) 49 IU/L (normal value <40); and SGPT (ALT) 52 IU/L (normal value <40). It also revealed a serum creatinine level (Cr) of 2.5 mg/dl (normal value 0.1-0.8) with a BUN of 42 mg/dl (normal value 6-18). His calculated creatinine clearance was 40 ml/min. He was noted to have a megaloblastic anemia with a hematocrit of 26%, hemoglobin of 8.8 gm/dl, MCV of 101.5 fL and a reticulocyte count of 4.2%. His serum vitamin B_{12} level was 451 pg/ml (150-1000) and his RBC folate level was 11.8 ng/ml (169-707). His serum iron level was 94 µg/dl; his ferritin level was 1199 ng/ml (normal value 12-250); and TIBC level was 313 µg/dl (normal value 250-420) being 30% saturated. His WBC count was 7700/mm^3 with 85% polymorphs. His serum ammonia (NH3) level was 50 µmol/L (normal value <33). Some of the other laboratory data obtained as part of his evaluation consisted of a serum protein electrophoresis (SPEP) documenting an albumin of 2.9 gm/dl (normal value 3.5-5.0); an elevated beta globulin fraction of 1.5 gm/dl (normal value 0.6-1.4); elevated IgA level of 446 gm/dl (normal value 54-374); a negative ANA; and a slightly elevated ceruloplasmin level of 44 mg/dl (normal value 16-29). His serum AFP level was normal at 3.2 ng/ml. All hepatitis serologic studies were negative (both hepatitis B and hepatitis C).

During his prolonged hospitalization, multiple other tests were performed including a CT scan of the abdomen which revealed a normal appearing liver with a volume of 1710 cc (normal 1200-1600 cc), and a slight enlargement of the pancreatic head without a definite mass being identified. This finding was not confirmed by an independent ultrasonographic examination of the pancreas. Both studies failed to reveal any evidence of biliary dilatation. A percutaneous liver biopsy revealed the presence of polymorphonuclear cells in the portal areas, central sclerosis, and subsinusoidal fibrosis. A repeat liver biopsy obtained 6 months later showed a similar picture with the additional finding of a mild lobular steatosis and portal area fibrosis. Hepatic iron and copper contents were 2, 250, and 125 µg/gm wet weight of liver respectively. An upper gastrointestinal endoscopy was normal. A colonoscopic examination including multiple random biopsies of the colon revealed a mild chronic inflammation and two small polyps which were removed and identified histopathologically as lipomas. Because of concern about the intensity and duration of the jaundice, two endoscopic retrograde cholangiopancreography (ERCP) procedures were performed six months apart. Both revealed normal pancreatic and biliary ducts. There was no evidence for primary sclerosing cholangitis (PSC) or pancreatic cancer.

Because of his encephalopathy, a neurologic examination consisting of head CT, EEG, and visual, auditory and somatosensory evoked potentials was obtained. They were all normal. Repeat EEG and brain stem evoked potential studies obtained six months later again because of continued clinical encephalopathy were reported as being normal. With cholestyramine and lactulose treatment, the patient's condition improved gradually over a 12-month period of observation.

At the end of 12 months of observation, the patient's total bilirubin level was 0.6 mg/dl; his alkaline phosphatase was 142 IU/L; his γ GTP was 20 IU/L; his SGOT (AST) was 21 IU/L; his SGPT (ALT) was 26 IU/L, and, his NH3 was 14 µmol/L.

Case 2.— A 33-year-old male weight lifter was referred to the authors with a three-week

history of jaundice, pruritus, and light stools. Five months prior to his admission, he started using a variety of anabolic steroids which included testosterone, stanozolol (17 methyl-2'W-5α-and rest-2-anal [3,2-c] pyrazol-17 BOL) and oxymetholone (17B hydroxy-2 [hydroxymethylene] 17-methyl-5αandrostam-3-one) to enhance his weight lifting potential. He also admitted to intermittently using cocaine. He denied any history of blood transfusions or hepatitis, but had been purposefully ingesting a protein-enriched diet to enhance muscle synthesis. His physical examination was abnormal only for jaundice and hepatomegaly.

Upon admission to hospital, the patient's total bilirubin was 44.3 mg/dl with a direct fraction of 40.7 mg/dl. His hematocrit was 51.6%, his WBC was 5400/mm^3, his BUN was 10 mg/dl, with a creatinine of 1.4 mg/dl. His serum ceruloplasmin was elevated at 64.3 mg/dl (normal value 16-29). Other studies included a serum albumin of 4.1 gm/dl, NH3 of 72 µmol/L, prothrombin time (PT) of 17.4 seconds, serum iron of 191 µg/dl, ferritin of 3044 ng/ml, and a TIBC of 479 µg/ml (40% saturated). His serum protein electrophoresis demonstrated a slightly elevated alpha-2 component which was 1.25 gm/dl. All autoimmune markers were negative. His serum AFP level was less than 20 ng/ml. All hepatitis B and C serology studies were negative.

A CT scan of the abdomen revealed an enlarged liver (3088 cc) with no intrahepatic bile duct dilatation. The pancreas was normal. An upper gastrointestinal endoscopy revealed one plus varices in the distal part of the esophagus. A colonoscopy was normal. Liver biopsy revealed evidence of a cholestatic hepatic injury with mild terminal perivesicular fibrosis. The hepatic lobular architecture was normal. The iron content of his liver was slightly increased at 1,552 µg per gram of tissue. An EEG was normal. After almost a month, the patient was discharged on cholestyramine and supplemental vitamin K.

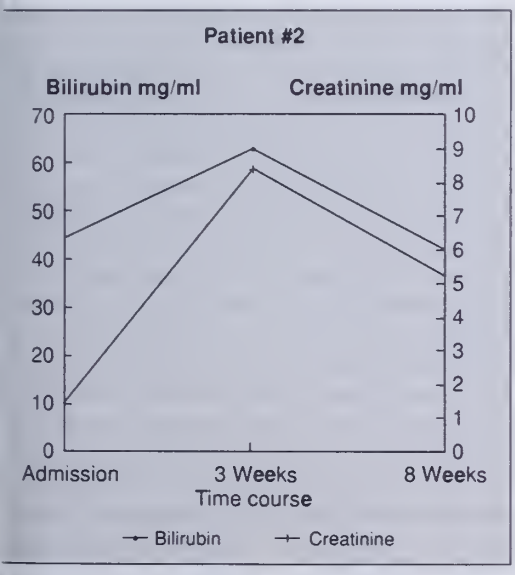

Figure 2. Serum bilirubin and creatinine levels in Case #2 shown at admission, 3 weeks and 8 weeks during the course of their illness.

Ten days after his discharge, the patient was readmitted with acute renal failure (ARF) manifested as a creatinine level of 8.5 mg/dl, BUN of 68 mg/dl, and total bilirubin of 62 mg/dl. His serum alkaline phosphatase level was 189 IU/L, γ glutamyl transpeptidase was 131 IU/L, SGOT (AST) was 34 IU/L, SGPT (ALT) was 12 IU/L and his serum ammonia level was 36 µmol/L. A renal biopsy revealed bile stasis and casts within the renal tubules, evidence of acute renal tubular necrosis and an acute tubulo-interstitial nephritis. The patient was treated with methylprednisone. After five additional weeks of hospitalization, he was discharged with a creatinine of 4 mg/dl, BUN of 48 mg/dl, total bilirubin of 41.6 mg/dl and a PT of 12.3 seconds. He slowly recovered normal renal and hepatic function over a 6-month period of observation without specific therapy except for the discontinuation of anabolic steroid use.

Case 3.—A 53-year-old male was admitted to the liver transplant service with a 2- week history of jaundice, nausea, vomiting, diarrhea, ascites, and pruritus. His prior history was pertinent for the use of testosterone 10 mg PO three times per day, for three months prior to his admission having been prescribed for him as a treatment for impotence. He denied any prior history of blood transfusions or excessive alcohol intake. His physical examination was pertinent only for jaundice. His admission blood work revealed a total bilirubin of 47.8 mg/dl with 45.6 mg/dl direct fraction, alkaline phosphatase of 294 IU/L, γ glutamyl transpeptidase of 85 IU/L, SGOT (AST) of 44 IU/L, SGPT (ALT) of 27 IU/L, NH3 of 42 µmol/L, BUN of 30 mg/dl, creatinine of 1.8 mg/dl, hematocrit of 52.3%, hemoglobin of 17.1 gm/dl, WBC of 7400/mm^3, MCV of 93.8 fL, PT of 11.9 seconds, albumin of 3.5 gm/dl, AFP level of 4 ng/ml, CEA level of 4.4 ng/ml, alpha-1-antitrypsin level of 207 mg/dl (normal value 85-213) and ceruloplasmin level of 65 mg/dl (normal value 16-29). Iron studies demonstrated a total iron of 147 µg/dl, more than 5000 ng/ml of ferritin, and TIBC of 207 µg/dl (44% saturation). His RBC folate level was 386 ng/ml (normal value 169-

Jaundice, when it occurs, does so usually one month or more after the initiation of the offending drug.

707) and his serum B_{12} level was 1263 pg/ml (normal value 150-1000). All autoimmune markers were negative. The patient's serum protein electrophoresis was entirely normal. His T4 level was 1.5 µg/dl (normal value 5-12) with a TSH of 0.91 µIU/ml (normal value 0.5-10) consistent with the diagnosis of euthyroid sick state and thyroid binding hormone deficiency. An abdominal CT revealed a fairly large liver with a computed volume of 2172 cc. It was homogeneous in appearance with no intrahepatic bile duct dilatation. Gall stones were present within the gallbladder. The portal vein was patent both by ultrasound and CT. An ERCP was normal with no intra- or extrahepatic bile duct abnormalities. Upper gastrointestinal endoscopy revealed kissing ulcers in the duodenal bulb associated with multiple other duodenal erosions. Except for a 4 mm colonic polyp, the colonic mucosa was normal. A hepatitis B serologic screen was positive for hepatitis B surface (IgG) and core antibodies.

The cholestasis associated with anabolic steroid use is often prolonged and renal dysfunction can occur in such cases.

A percutaneous liver biopsy revealed acute pericholangitis with marked cholestasis and bile plugs. A combined caffeine/antipyrine clearance test obtained at two different time periods separated by 3 months was normal on both occasions. A diagnosis of testosterone-induced cholestasis was made and the patient was discharged on ursodeoxycholic acid with a total bilirubin level of 46 mg/dl with the anticipation that it would take six to twelve months to achieve complete resolution of his jaundice. Three weeks later, the patient was readmitted with renal insufficiency and encephalopathy having a serum creatinine level of 8.2 mg/dl and BUN of 98 mg/dl. His serum total bilirubin level was still elevated at 47 mg/dl. His other studies included a PT of 15.5 seconds, albumin of 2.7 gm/dl, NH3 of 86 µmol/L, hematocrit of 33%, hemoglobin of 12 gm/dl, MCV of 87 fL, and urine analysis which was positive only for granular casts. His calculated creatinine clearance was 18 ml/min. A renal plasma flow scan revealed a low normal filtration rate. Eventually, with intensive medical care, both renal and hepatic function improved. The patient left the hospital the second time with a total bilirubin of 7.8 mg/dl and serum creatinine of 2.8 mg/dl on ursodeoxycholic acid and lactulose. After six additional months of observation, his serum, renal, and hepatic parameters were all normal.

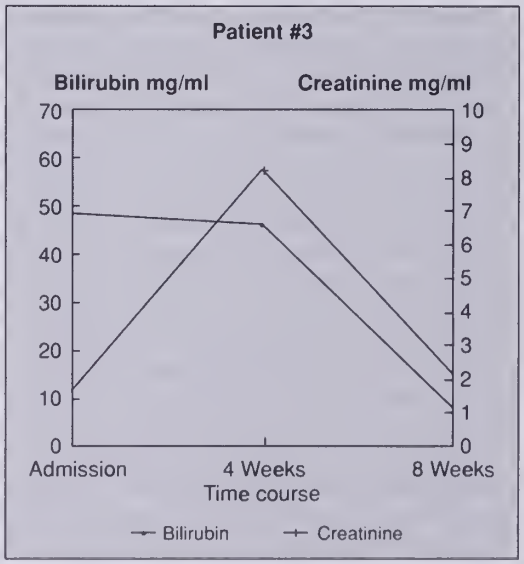

Figure 3. Serum bilirubin and creatinine levels in Case #3 shown at admission, 4 weeks and 8 weeks during thee course of their illness.

Case 4. —A previously healthy 26-year-old competitive power lifter was referred because of generalized malaise, nausea, vomiting, and increasing jaundice occurring over a four-week period. For competitive reasons, he had been taking stanozolol 125 mg intramuscularly twice weekly for a one-month period prior to the onset of the jaundice. His past medical history was unremarkable and was free of any history of preexisting liver or kidney disease. He was not taking any other drugs and did not smoke, consume ethanol, or use street drugs. Physical examination revealed an exceptionally muscular white man with scleral icterus and a mildly large tender liver that was 8 cm below the costal margin. There were no extrahepatic manifestations of liver disease or evidence of connective tissue disease. The remainder of his physical examination was unremarkable. Initial laboratory data revealed normal hematological parameters except for the presence of target cells in the peripheral smear, a prothrombin time (PT) of 12.5 s (<13.5), normal serum electrolytes, AST of 33 U/L and urea of 19.0 mg/dl. The serum creatinine nine level was abnormal at 1.8 mg/dl, as was the alkaline phosphatase level of 137 U/L, and a total bilirubin level of 19.5 mg/dl. It was felt that the patient's jaundice was the result of anabolic steroid ingestion, and he was returned to the care of his family physician.

One month later, the patient was readmitted because of increasing jaundice, persistent vomiting, and new onset renal failure. He had experienced an 18-pound weight loss over the preceding one month. Repeat laboratory examination revealed jaundice, normal hematologic parameters, a normal PT and serum electrolytes, a BUN of 29.4 mg/dl, a creatinine of 4.7 mg/dl, an alkaline phosphatase of 369 U/L, an albumin of

2.5 mg/dl, a calcium of 9.4 mg/dl, a phosphorous of 6.5 mg/dl, and a normal serum creatinine kinase (CK) value. Viral hepatitis serologies (both HBV and HCV) and an antinuclear antibody (ANA) were negative. A urinalysis revealed 10-25 leukocytes for HPF but no casts. A 24-hour urine was negative for protein. Chest radiographs were negative, as was an abdominal ultrasound which demonstrated normal kidney size and no evidence of extrahepatic biliary obstruction.

A percutaneous liver biopsy revealed marked central lobular cholestasis, feathery hepatocyte degeneration, and pericentral fibrosis; the hepatic architecture was preserved. Mild periportal fibrosis was evident but without linkage of adjacent portal tracts. The findings were interpreted as being consistent with anabolic steroid-induced cholestasis. A renal biopsy demonstrated a single focus of mononuclear cells consistent with a possible interstitial nephritis. There were no glomerular changes on either light or electron microscopy; all immunofluorescent studies were unremarkable.

The patient was provided with supportive medical treatment and gradually improved. On discharge, two months after having discontinued the stanozolol, the patient's alkaline phosphatase level was 315 U/L, total bilirubin was 24 mg/dl, and serum creatinine was 3 mg/dl. Serial laboratory data revealed a progressive decline in the patient's hepatic enzymes, all of which returned to normal values after five months of stanozolol abstinence. The patient recovered with mild residual chronic renal injury, having a stable serum creatinine level of 1.6 mg/dl.

Discussion

When used in therapeutic doses, anabolic steroid produces clinical jaundice in only a small number of recipients. In contrast, a large number of recipients can be shown to develop a mild degree of hepatic dysfunction. Jaundice, when it occurs, does so usually one month or more after the initiation of the offending drug. Moreover, its intensity appears to be dose dependent. Malaise, anorexia, and nausea usually precede the jaundice. Pruritus can be severe and often is the principal reason why patients seek medical advice. In most patients with clinical jaundice, the laboratory studies reveal a peak total bilirubin of 10 mg/dl or more. To date, the highest peak bilirubin level reported following anabolic steroid use has been 34 mg/dl.[5] Resolution of anabolic steroid-induced jaundice is slow and typically requires weeks to months to clear. Death is extremely rare but has been reported.[3]

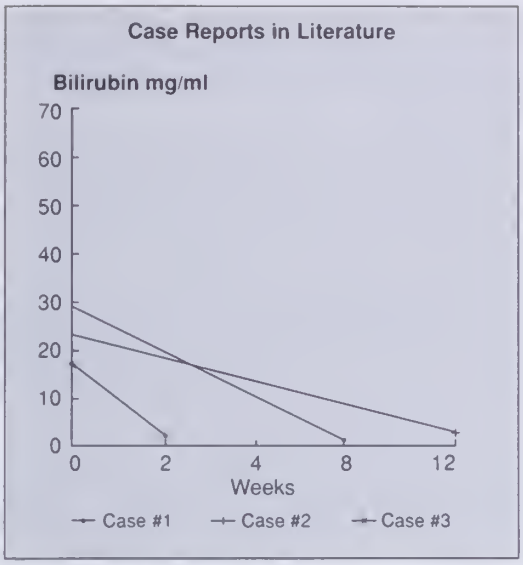

Figure 4. Serum bilirubin and creatinine levels in 3 case reports from the literature shown at admission and 8 weeks, admission and 12 weeks, and admissionand 2 weeks, respectively.

As reported in the literature,[5,6] the cases herein reported with androgen-induced cholestasis presented with a history of nausea, vomiting, jaundice, weight loss, and pruritus occurring within three to four months of starting steroid therapy. With steroid withdrawal and extensive medical care, complete resolution of all identifiable hepatic and renal dysfunction occurred in each case. In contrast to all prior cases, renal dysfunction was noted in three of the four present cases. Common hepatic histologic findings in the 4 cases were the presence of a pericholangitic acute inflammatory picture with preservation of the lobular architecture and absence of hepatocellular cellular necrosis.[7]

The presence of a normal biliary system by ultrasound, CT and ERCP as well as near normal or normal levels of all canalicular enzymes were all important in resolving the differential diagnosis in these four cases. Interestingly, the serum alkaline phosphatase level is reported to be double the upper limit for normal values in half the cases and more than triple the upper limits of normal in 5% of the cases reported in the literature.

The mechanisms by which exogenous androgens induce cholestasis are not fully known. It is likely that they inhibit the Na+, K+-ATPase activity of hepatocytes by altering the fluidity of the liver cell membrane. In addition, androgens also reduce hepatic bile acid uptake and bile flow.[9] The structural resemblance of anabolic steroids

to bile acids raises the possibility that they may compete for bile acid receptors and impair the uptake and excretion of bile acids at very high plasma levels.

The important clinical points to be gleaned from these four cases is that the cholestasis associated with anabolic steroid use is often prolonged (6 months or more) and that renal dysfunction can occur in such cases. Although not reported specifically in the past, renal dysfunction occurring in cases of anabolic steroid cholestasis may be more common than previously appreciated. Whether the renal dysfunction seen in such cases can be prevented with careful attention to issues of hydration and the use of both IV contrast agents and aminoglycoside antibiotics in such cases is unclear. Nonetheless, caution relative to such issues in cases of anabolic steroid-associated jaundice would appear to be appropriate.

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The Authors

Ahmet Gurakar, MD, is a fellow in gastroenterology at the University of Texas, Galveston. Paolo Caraceni, MD, is a research fellow at the Oklahoma Transplantation Institute (OTI), Baptist Medical Center of Oklahoma, Oklahoma City. Stefano Fagiuoli, MD, is a clinical fellow in transplantation medicine at OTI. David H. Van Thiel, MD, is chief of transplantation medicine and director of the Liver and Digestive Disease Center at OTI.

Autologous Bone Marrow Transplantation for Metastatic Breast Cancer

Ruben A. Saez, MD; George B. Selby, MD; R. Bradley Slease, MD; Robert B. Epstein, MD; Romeo A. Mandanas, MD; Dennis L. Confer, MD

Metastatic breast cancer accounts for 18% of cancer deaths among women in the U.S. Conventional combination chemotherapy produces responses in 50% to 80% of women with metastatic breast cancer, but is never curative. A major medical center administered high-dose chemotherapy and autologous bone marrow transplantation to 68 women with metastatic breast cancer between 1983 and 1993. Forty-nine of these women had estrogen receptor-negative tumors, a poor prognostic sign. Eighteen women with estrogen receptor-positive tumors had failed prior hormonal manipulation or had metastatic breast cancer at initial diagnosis. Prior to transplantation, 37 women were in first complete or partial remission, 8 were in their second complete or partial remission, 4 had stable disease, 14 had progressive disease, and 5 were in untreated relapse. Bone marrow transplantation preparatory regimens included high doses of single agents or combination chemotherapy.

Among women not in remission before transplantation, 71% entered a partial or complete remission following transplantation. Overall, 29 women (43%) were in complete remission after marrow transplantation. Twelve women (18% overall) remain free of disease between 2 and 73+ months post-ABMT. Those in first complete or partial remission prior to transplant (37 patients) had a higher response rate (86%) and higher complete responses (62%), and 10 (27%) are free of disease.

There were nine treatment-related deaths (13%). Forty-seven patients (69%) have died from breast cancer following autologous transplantation. Relapses occurred primarily at sites of previous disease. All relapses have occurred within 22 months of ABMT.

Conclusion: Autologous bone marrow transplantation for metastatic breast cancer in first complete or partial remission has produced a 27% disease-free survival. This therapy should be considered for selected patients with metastatic breast cancer.

BREAST cancer is the most common malignancy among women in the U.S. and accounts for 18% of all cancer deaths, second only to lung cancer.[1] While breast cancer is highly curable when detected in early stages, 40% of women who present with lymph node involvement and 20% of those with node-negative presentations will relapse and die of their disease.[2] Conventional combination chemotherapy achieves responses in 50% to 80% of patients with metastatic breast cancer, including 10% to 20% complete responses.[3-5] Unfortunately, the median duration of remission is only 12 months and median survival is 24 months. Few patients are alive at five

From the University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, and the Medical Arts Pavilion, Newark, Delaware.

Direct correspondence to Ruben A. Saez, MD, University of Oklahoma Health Sciences Center, P.O. Box 26901, Oklahoma City, Oklahoma 73190.

years, and no one is cured. Survival and response duration are worse for women with one or more of the following: estrogen receptor (ER) and progesterone receptor (PgR) negative tumors, recurrence within 12 months of initial diagnosis, more than one site of metastatic disease, and any visceral metastasis. Among patients with these poor prognosis features, survival at three years is less than 10%.[6]

There is clinical evidence that responses to chemotherapy correlate with the dose intensity of treatment. However, the escalation of drug doses is limited by marrow toxicity.[7-9] Protection against this toxicity can be achieved with the infusion of the patient's own bone marrow, which has been cryopreserved prior to the initiation of intensive chemotherapy. With such autologous bone marrow transplantation (ABMT) techniques, hematologic toxicity is averted and three- to tenfold dose escalation is possible, limited only by toxicity to non-marrow organs.[10,11]

Since 1983 the bone marrow transplant service at the medical center has performed 68 transplants for women with metastatic breast cancer. This paper details the results and future plans.

Patients and Methods

Sixty-eight women with metastatic breast cancer aged 27 to 59 years (median 41 years) were enrolled in one of four consecutive Phase II trials evaluating ABMT in malignant diseases. There were six patients treated with cyclophosphamide alone at 150-200 mg/kg (Cy), 28 with cyclophosphamide at 160-200mg/kg and BCNU at 600-900mg/M^2 (CyBCNU), three with cyclophosphamide at 160mg/kg, etoposide at 1200mg/M^2, cisplatin at 100mg/M^2, and BCNU at 600mg/M^2 (CEBP), and 31 with cyclophosphamide at 120-160mg/kg, etoposide at 1200-1800mg/M^2, carboplatin 1600-2000mg/M^2 (CEC).

All the patients transplanted had characteristics associated with shortened survival: ER-negative tumors, ER-positive tumors failing prior hormonal manipulation or metastatic disease at diagnosis regardless of receptor status. In the early years of the program patients responding to conventional chemotherapy (chemosensitive), as well as those refractory to these treatments (chemoresistant), were transplanted, while recently only those with chemosensitive disease have been treated. All patients had adequate performance status (SWOG grade 0-2) and organ function.

Pretransplant clinical characteristics are listed in Table 1. Forty-nine patients (72%) had ER-negative tumors, 18 (26%) had ER-positive tumors, and in one (2%) the ER assay was not done. Twenty patients had evidence of metastatic disease at initial diagnosis (eight had inflammatory or locally advanced breast cancer), while 48 had suffered relapse following initial therapy undertaken with curative intent. Of these 48, 16 had relapsed within 12 months of initial therapy, 15 had relapsed between 12 and 24 months, and 17 had relapsed more than 24 months from diagnosis. Thirty-four of these 48 women (71%) had received adjuvant therapy: 25 received chemotherapy, 3 tamoxifen, and 6 received both.

Sites of metastatic disease included lung ± other sites (22 patients, 32%), liver ± other sites (15 patients, 22%), soft tissue only (15 patients, 22%), bone or bone marrow ± soft tissue (14 patients, 21%), and brain ± soft tissue (2 patients, 3%). Prior to transplantation, most patients had received chemotherapy for their metastatic disease (93%), usually adriamycin-based chemotherapy (92%) and usually only one regimen (90%).

Twenty-two patients (32%) received radiotherapy to the chest wall or to painful bone metastases. Disease status pre-ABMT included first complete remission (CR1, 15 patients), or first partial remission (PR1, 22 patients), second or greater complete or partial remission (≥CR2/PR2, 8 patients), stable disease (SD, 4 patients), pro-

Table 1. Pre-Transplant Clinical Characteristics
n = 68

Age (years	
Median	41
Range	27-59
Receptors Status (ER/PgR)	
Negative	49 (72%)
Positive	18 (26%)
Unknown	1 (2%)
Prior Adjuvant Therapy	34 of 48 (71%)*
Sites of Metastases	
Lung ± other	22 (32%)
Liver ± other	15 (22%)
Soft tissue only	15 (22%)
Bone/Bone Marrow ± soft tissue	14 (21%)
Brain ± soft tissue	2 (3%)
Prior Radiotherapy	22 of 68 (32%)
Disease Status Pre-ABMT	
CR1	15
PR1	22
≥CR2/PR2	8
SD	4
PD	14
Untreated	5

CR — Complete remission, PR — Partial remission, SD — Stable disease, PD — Progressive disease
*Percentage calculation excludes 20 patients who had metastatic disease at diagnosis.

gressive disease (PD, 14 patients), and untreated relapse (5 patients).

Bone marrow was collected from the iliac crests after bilateral biopsies had demonstrated adequate cellularity and the absence of tumor contamination. Details of the collection procedure and marrow processing have been described previously.[11] Before 1987, patients were admitted to private rooms on the oncology floor and, since 1987, to the bone marrow transplant unit; all were managed with mask and hand washing isolation techniques. Patients received high dose chemotherapy according to one of the four preparatory regimens detailed above and received autologous bone marrow on Day 0. A mean of 2.28×10^8 nucleated autologous marrow cells/kg (range 0.91-6.28) was infused.

All patients had double or triple lumen silastic central venous catheters. Antimicrobial prophylaxis has evolved over the years, but most patients received trimethoprim/sulfamethoxazole for antipneumocystis prophylaxis, quinalones for antibacterial prophylaxis, acyclovir for positive herpes simplex serology and, since 1991, fluconazole for antifungal prophylaxis. Patients received standardized therapy for febrile neutropenia with broad spectrum antibiotics, and amphotericin B for persistent fever. Most patients received total parenteral nutrition and blood component support. Blood products were filtered to reduce leukocyte content and irradiated.

After hematologic recovery, full restaging was performed; residual disease was resected, if possible, and/or irradiated. Complete remission (CR) was defined as the disappearance of all tumor by physical exam and by scans; partial remission (PR) required at least 50% reduction in measurable lesions. For patients with bone disease only, improvement in the bone scan was considered a PR, while CR required healing on plain films or normalization of bone scan.

A response to marrow transplantation was defined as an improvement in the patient's disease status (for example, PR to CR or SD/PD to PR or CR); thus, women in CR before transplantation could not be evaluated for response. Following transplantation, freedom from progression (FFP) was defined as the absence of relapse (for women in CR post-transplantation) or the absence of progression (for women in PR post-transplantation). Life table estimates of progression-free survival were calculated by the method of Kaplan and Meier,[12] measured from the date of ABMT to the date of relapse, progression or death. Survival curves were compared by the Mantel-Haenszel statistic.[13]

Results

Engraftment.— The median time to recovery to 500 neutrophils per µl was 15 days (range 11-35) and to a platelet count ≥ 50,000 per µl was 24 days (range 9-67). Nine patients, who died from treatment-related toxicity, were not evaluable for engraftment; 4 additional patients could not be evaluated for platelet engraftment because of death from progressive breast cancer (3) or cytomegalovirus infection (1).

Toxicity.—A detailed analysis of transplant-related toxicity is beyond the scope of this report, but clearly the multi-agent BMT regimens were more toxic than high-dose cyclophosphamide alone. Regimens containing BCNU (CyBCNU and CEBP) had as major toxicities venoocclusive disease of the liver (VOD) and diffuse alveolar hemorrhage (DAH), while regimens containing carboplatin (CEC) were complicated primarily by renal toxicity and enterocolitis.

There were nine regimen-related deaths (13%) from the following causes: VOD (2), respiratory failure (3), bacterial sepsis (1), fungal sepsis (2), and intracranial hemorrhage (1). Factors that appeared to increase the risk of toxic death include older age (two of two patients over age 55), extensive liver metastases (four deaths), and obesity (two deaths in patients whose weight was more than twice their ideal body weight).

Outcome.— Overall, autologous bone marrow transplantation produced a 71% response rate and 43% of women (29 of 68) were in complete remission following the procedure (Fig. 1). At last follow-up in January 1994, 12 women (18%) remained free from progression between 2 and 73+ months (median 25 months) following ABMT. Survival of patients who failed to respond to transplantation was very short.

Women responding to conventional chemotherapy at first evidence of metastatic disease (ie, those transplanted in first complete remission or first partial remission) had the best outcome: 23 of 37 (62%) were in CR post-ABMT and 10 (27%) are FFP with a median follow-up of 26 months (Fig. 1). The overall outcomes for these patients are significantly better than those of patients transplanted with other categories of disease (≥CR2/PR2, SD, PD or untreated, Table 2).

Forty-one women (60%) have relapsed or progressed at a median of 7 months post-ABMT (range 3-22 months). Relapses have occurred primarily at sites previously involved with breast cancer, suggesting that failure to eradicate bulky disease with ABMT is the cause of relapse, rather than infusion of occult tumor cells with the marrow innoculum. In particular, only 8% of patients

Overall, autologous bone marrow transplantation produced a 71% response rate and 43% of women (29 of 68) were in complete remission following the procedure.

with liver, lung, or two or more bone metastases are free from recurrence. Although the clinical course after relapse is variable, the median survival is only 5 months (range 0-34 months). Patients with hormone receptor-negative tumors have tended to relapse earlier post-ABMT than those with receptor-positive tumors (median time to relapse of 6 months vs. 12 months), but the median survival of both groups after relapse is similar. There is currently one patient alive with disease at 7 months after relapse from ABMT.

Figure 2 presents the Kaplan-Meier estimates of progression-free survival for the 68 patients who underwent ABMT at our institution. At 24 months, the estimated progression-free survival for patients transplanted in CR1 or PR1 has plateaued at 21% (Fig. 2). Because no relapse has occurred more than 22 months after transplantation, these women are not only disease-free, but some may in fact be cured. In contrast, no patient transplanted with more advanced disease has reached the 24-month milestone (Fig. 2). The progression-free survival for women transplanted in CR1 or PR1 is significantly better by the Mantel-Haenszel statistic when compared to all others ($p<0.001$).

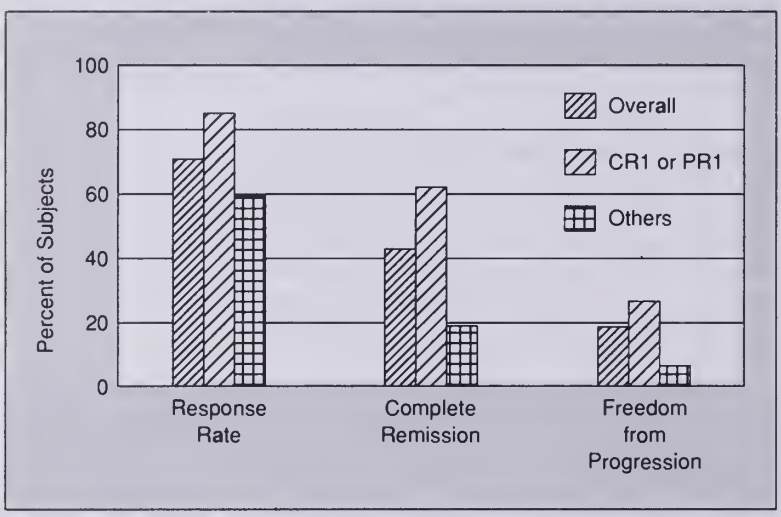

Figure 1. Response rate, complete remissions and freedom from progression for 68 women with metastatic breast cancer receiving autologous marrow transplantation between 1983 and 1993. Results are presented for the entire group (Overall) and for two subgroups: those transplanted in first complete or partial remission (CR1 OR PR1) and all others (Others, includes ≥CR2/PR2, stable disease, progressive disease and untreated relapse).

Table 2. Response and Outcome of Autologous Marrow Transplantation According to Pretransplantation Status

		Response		Outcome in followup	
	n	CR	NR/TD	Relapse	FFP
CR1/PR1	37	23	3	24	10
		p<.001	p<.05	p=NS	p<.05
Others	31	6	12	17	2

CR — Complete remission, NR/TD — No response or Toxic death, FFP — Freedom from progression, CR1/PR1 — first complete or partial remission. Patients not listed in the response column are those who achieved a PR. Patients not listed in the outcome column are those who had no response or had toxic death. All p-values are Chi-square comparisons.

Discussion

Women with hormonally resistant metastatic breast cancer have a dismal prognosis with a median survival of less than 2 years, and less than 10% survival at 5 years.[4] Conventional chemotherapy is palliative in intent. The clinical evidence of a dose-response effect in breast cancer has encouraged investigations of high dose chemotherapy with bone marrow rescue in these patients. Initially single agents were used, and while responses were seen, they were not complete, and complete response is the *sine qua non* for benefit in cancer treatment. Combinations of agents in high doses have been tried with the attainment of complete responses, but with increased toxicity.[11,14]

Over the past ten years, our medical center has performed 68 autologous bone marrow transplants for women with metastatic breast cancer. The criteria used for the selection of patients and the transplantation regimens utilized have evolved during this time. Initially, this treatment was considered for any woman with metastatic breast cancer, including those with progressive and refractory disease; our initial preparative regimen used only a single high-dose agent. Subsequently, high-dose chemotherapeutic agents were combined in the transplant regimens and the selection criteria have been refined to exclude women with advanced, refractory disease. Our early trials showed that women who fail to achieve an objective tumor response pre-ABMT are highly unlikely to benefit from transplantation. Even those who achieve a response at a second or greater relapse (≥CR2/PR2) do not seem to have lasting benefit; however, the numbers are small and more cases in this category should be studied.

Among the women who benefit the most from autologous transplantation, i.e., those at first sign of metastatic disease who are responding to standard chemotherapy, 27% are alive without recurrence between 2 and 73+ months post-ABMT. These results are very encouraging; because all

relapses in our series have occurred within the first 22 months following transplant, some of these patients may be cured. The projected Kaplan-Meier survival of 21% represents a two- to three-fold improvement over the reported series with conventional chemotherapy in metastatic breast cancer, where <10% of hormonally refractory patients are alive at 2 years.[5]

There are still many improvements to be made: 60% of responses post-ABMT are transient with progression usually occurring at previous sites suggesting lack of eradication of existing tumor as the main reason for failure. While seeding of previous tumor sites by infusion of occult tumor cells in the marrow inoculum is a theoretical possibility, proof is lacking. For patients who relapse after transplant the outcome is poor, and half die within 5 months.

What to do for the future? Women with breast cancer who relapse at multiple sites or with resistant disease have derived little benefit from autologous transplantation in our series. Because of this, we have undertaken to identify women at initial diagnosis who are at high risk for relapse and offer ABMT as adjuvant therapy before metastases occur. Since 1990 we have treated 21 women with more than ten axillary nodes positive and/or inflammatory breast cancer with conventional chemotherapy followed by ABMT.[15] With a median followup of 20 months from diagnosis (14 months from ABMT), 92% of these subjects remain free from progression. These are very encouraging results and we plan to continue this approach.

Unfortunately, many patients with fewer than ten positive nodes, or with node-negative disease at diagnosis, who are not candidates for adjuvant marrow transplantation will nevertheless relapse, and they may become candidates for transplantation at that time (Table 3). Women with a single site of metastatic disease who attain first complete or partial remission (CR1/PR1) should receive autologous marrow transplantation. For women with responsive disease (CR1/PR1) and more than a single site of metastasis, we have opened a double transplantation protocol, i.e., two cycles of high-dose therapy with marrow progenitor cell support, in an effort to decrease post-transplantation relapses. Patients undergo two consecutive hospitalizations, four to eight weeks apart, and receive two different high dose chemotherapy regimens. To diminish toxicity, shorten the hospital stay, and decrease total costs, this protocol employs peripheral blood stem cells in addition to autologous bone marrow.

Women with metastatic breast cancer who lack an objective response (SD) or who progress (PD) with conventional chemotherapy, and even those with responding disease but at a later relapse (≥CR2/PR2), are unlikely to benefit from our current marrow transplantation protocols. These patients will be better served by enrollment in Phase I-II trials looking at new chemotherapeutic agents or in new Phase I-II ABMT studies.

The results at this medical center with autol-

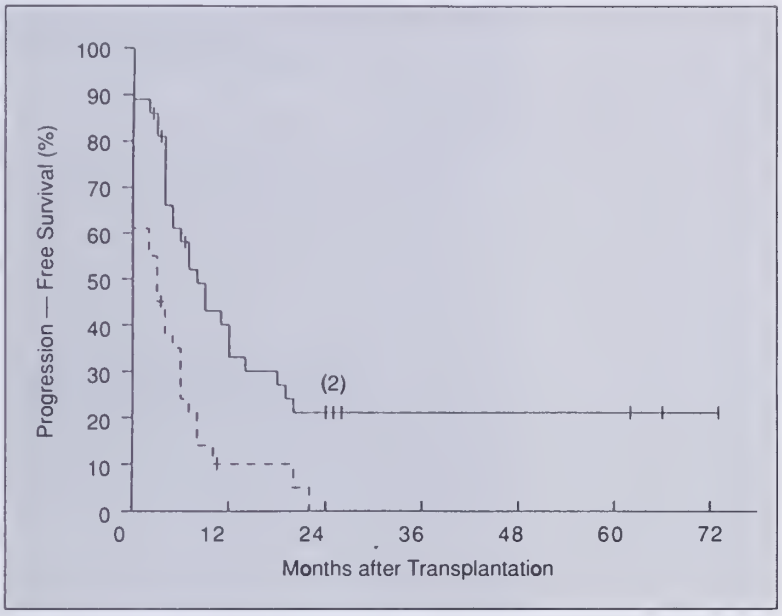

Figure 2. Kaplan-Meier estimated progression-free survival for 68 women receiving autologous marrow transplantation for metastatic breast cancer. Women in first complete or partial remission (——) are compared to all others (- - -), which includes ≥CR2/PR2, stable disease, progressive disease and untreated relapse. The two curves are significantly different by the Mantel-Haenszel statistic, p<0.001. Vertical bars (|) denote progression-free survivors, the numeral above the bar indicates multiple survivors at a single time point.

Table 3. Autologous Bone Marrow Transplantation Approach to Breast Cancer

Patients	Treatment
1. High risk (non-metastatic) breast cancer – Inflammatory/locally advanced ≥ 10 axillary nodes positive	ABMT as adjuvant
2. Metastatic Breast Cancer – CR1/PR1	
1 Metastatic site	ABMT × 1
≥ 2 Metastatic sites	ABMT × 2
≥CR2/PR2	ABMT – New Regimens

CR — Complete Remission
PR — Partial Remission
ABMT × 2 — Two consecutive transplants as described in the text

ogous bone marrow transplantation for metastatic breast cancer are similar to those of other centers[16,17] and suggest that aggressive therapy may cure some patients with metastatic breast cancer. While controlled prospective randomized studies are in progress, with the data available in 1994, we consider ABMT for selected patients with hormone-resistant metastatic breast cancer to be the best available treatment.

Acknowledgments

The authors wish to thank Carroll Brandt for preparation of the manuscript and Dr. J. George for helpful comments. We also acknowledge the Scanlan Marrow Transplant Fund for support of this research.

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The Authors

Ruben A. Saez, MD, is an assistant professor of medicine at the University of Oklahoma Health Sciences Center (OUHSC) in Oklahoma City and associate director of bone marrow transplantation.

George B. Selby, MD, is an associate professor of medicine at OUHSC and assistant chief of the medical service at the Veterans Administration Medical Center of Oklahoma in Oklahoma City.

R. Bradley Slease, MD, is a clinical professor of medicine at the Jefferson Medical College of Thomas Jefferson University, Philadelphia, Pa.

Robert B. Epstein, MD, is Eason Professor of Medicine at OUHSC.

Romeo A. Mandanas, MD, is an assistant professor of medicine at OUHSC.

Dennis L. Confer, MD, is an associate professor of medicine and director of marrow transplantation at OUHSC and medical director of the National Marrow Donor Program, Minneapolis, Minn.

Understanding Mechanical Ventilation: Patient Monitoring, Complications, and Weaning

Brent R. Brown, MD

This article is the second of a two-part series reviewing the fundamental concepts of mechanical ventilation. The techniques of monitoring patients receiving mechanical ventilation and the potential complications of mechanical ventilation are discussed. Techniques for weaning from mechanical ventilation are reviewed and guidelines are provided concerning patient suitability for extubation.

An article published last month in this journal reviewed the indications for and basic modes of mechanical ventilation, and provided management guidelines for the mechanical ventilation of adults.

Patient Monitoring During Mechanical Ventilation

Monitoring during mechanical ventilation allows the clinician to follow the course of the disease process and the patient's response to therapy. Additionally, monitoring techniques (Table 1) may provide early detection of complications providing the physician with the opportunity to intervene. The bedside examination is one form

Table 1. Monitoring During Mechanical Ventilation

Parameter Monitored	Monitoring Technique
Bedside Findings	The five senses
Oxygenation	Arterial O_2 tension (PaO_2) Mixed venous O_2 tension ($P_{\bar{v}}O_2$) O_2 saturation by co-oximetry Pulse oximetry
Ventilation	Arterial CO_2 tension (PCO_2) Expired minute ventilation ($\dot{V}_E$) End tidal CO_2 measurement
Hemodynamics	Automated non-invasive blood pressure monitors Arterial line Pulmonary artery catheter
Pulmonary Mechanics	Static compliance Peak airway pressure Plateau (static) airway pressure
Ventilator Function	Ventilator alarms systems

Direct correspondence to Brent R. Brown, MD, University of Oklahoma Health Sciences Center, Department of Medicine, Post Office Box 26901, Room 3SP 400, Oklahoma City, OK 73190.

Table 2. Important Bedside Findings in Mechanically Ventilated Patients
Patient-ventilator dysynchrony
Patient agitation
Tachypnea
Breathing pattern
Use of accessory muscles of respiration
Supraclavicular/intercostal retractions
Cyanosis
Subcutaneous emphysema
Breath sounds
Heart sounds

The adequacy of ventilation is primarily monitored by measurements of arterial CO_2 tension ($PaCO_2$).

of monitoring often underutilized in the intensive care unit. Mechanically ventilated patients are difficult to examine due to their immobility and the presence of numerous devices at their bedsides. A result has been excessive physician reliance on artificially derived data and a neglect of bedside findings in critically ill patients. The seasoned clinician can obtain information unmeasurable by machines (Table 2) and the importance of this information in monitoring mechanically ventilated patients has recently been emphasized.[1] Some bedside findings are closely associated with specific problems. For example, subcutaneous emphysema of the neck strongly suggests the presence of pneumomediastinum. Most physical findings are less specific but indicate the need for further investigation for potential problems. Tachypnea, retractions, and accessory muscle use during mechanical ventilation may result from hypoxemia or hypercapnia. Patient-ventilator asynchrony may reflect the need for adjustments in tidal volume, inspiratory flow rate, triggering sensitivity, or percentage of inspired oxygen.

Although they will never supplant the physical examination, a number of technology-dependent monitoring techniques have become standard practice for mechanically ventilated patients. Measurements of the arterial oxygen tension (PaO_2) are routinely made and calculations of the arterial oxygen saturation (SaO_2) can be provided based on the PaO_2. Knowledge of the SaO_2 is important, as the SaO_2 is more significant than the PaO_2 in determining the oxygen (O_2) content of the blood and the transport of O_2 to the tissues. *Calculated* values of SaO_2 generally correlate well with directly *measured* values. However, *calculated* values of SaO_2 do not account for shifts in the oxyhemoglobin dissociation curve or for elevations in carboxyhemoglobin or methemoglobin which sometime accompany critical illness. Co-oximetry directly *measures* the SaO_2, and daily measurements of SaO_2 are useful in critically ill patients with marginal oxygenation to confirm that calculated SaO_2 values approximate measured values.

Pulse oximetry is useful in following trends in oxygenation and may provide early warning of deteriorations in gas exchange. Pulse oximetry lessens the frequency in which arterial blood gases (ABGs) must be measured. A saturation greater than 92% by pulse oximetry generally correlates well with a normal *measured* SaO_2. Poor skin perfusion, elevations in carboxy or methemoglobin levels, and dark skin pigmentation lessen the accuracy of pulse oximetry.[2] In critically ill patients, daily measurements of SaO_2 should be obtained to ensure oximetric saturations approximate true values.

The adequacy of ventilation is primarily monitored by measurements of arterial CO_2 tension ($PaCO_2$). The expired minute ventilation (V_E) is the product of the respiratory rate and the tidal volume (V_T) and is also useful in following the patient's ventilatory status. Serial measurements of V_E should be recorded at the patient's bedside. Increases in V_E may reflect hypoxemia or the development of metabolic acidosis. Sudden decreases in V_E can be caused by reductions in respiratory drive or the development of leaks in the ventilator circuitry. End tidal CO_2 monitoring is extensively used intraoperatively to monitor the adequacy of ventilation but is inaccurate in the setting of parenchymal lung disease, greatly limiting its utility in the critical care setting.[3]

Hemodynamic monitoring with arterial lines and pulmonary artery catheters is not mandatory during mechanical ventilation, but is helpful for hemodynamically unstable patients and for those requiring positive end expiratory pressure (PEEP) levels which might compromise cardiac output ($\geq$10 cm H_2O). The pulmonary artery catheter allows measurement of mixed venous blood gases, of cardiac output, and of pulmonary capillary wedge pressure. In most situations the mixed venous O_2 saturation (S_VO_2) reflects the adequacy of tissue O_2 delivery, as tissues extract a higher percentage of delivered O_2 as total O_2 delivery falls. Normal resting values of S_VO_2 are 73% to 85%. Values below 50% indicate critically low tissue O_2 delivery. A reduction in S_VO_2 can be due to an increase in O_2 consumption or to a reduction in cardiac output, arterial oxygenation, or hemoglobin level.

Table 3. Complications Increasing Peak but Not Plateau Pressure
Bronchospasm
Mucosal edema
Kinked endotrachael tube
Partial endotracheal tube obstruction by secretions

Table 4. Complications Increasing Peak and Plateau Pressures
Pulmonary edema
Lobar atelectasis or consolidation
Intubation of mainstem bronchus
Tension pneumothorax
Secretion obstruction of major airway
Auto-PEEP

Several easily measured parameters of pulmonary mechanics are useful in following the course of a patient's illness and in evaluating acute decompensations of mechanically ventilated patients. *Compliance* of the respiratory system describes the relationship between lung volume and airway pressure. Static compliance (C_S) can be calculated by using the formula:

$$C_S = \frac{\text{tidal volume (exhaled)}}{\text{plateau pressure-PEEP}}$$

The patient should be relaxed, allowing for passive lung inflation in order to obtain accurate estimates of C_S. Normal values of C_S are 80-100 ml/cm H_2O. Serial determinations of C_S are useful in following the course of ARDS patients, who have marked reductions in C_S. Acute reductions in C_S can occur in patients who develop pulmonary edema, atelectasis, endotracheal tube migration into a mainstem bronchus, or pneumothorax.

Measurements of peak and plateau airway pressure are easily made and should be recorded at the bedside for ready reference. Plateau pressure can be measured by adding an inspiratory pause to the respiratory cycle, by pushing an "inspiratory hold" button, or by briefly occluding the expiratory port of the circuit, depending on the type of ventilator used. The difference between peak and plateau pressure should be no greater than 10 cm H_2O when an endotracheal tube (ET) of adequate diameter is used ($\geq$7.5 mm).[4] Peak airway pressure is effected by changes in airway resistance *and* respiratory system compliance, whereas changes in plateau pressure primarily reflect changes in compliance alone. The difference between the peak and plateau pressure is useful in following the course of patients with bronchospasm. Airway resistance declines as bronchospasm subsides, lessening the difference between peak and plateau pressure. Some complications of mechanical ventilation increase airway resistance, thus increasing peak airway pressure but not plateau pressure (Table 3). Other factors reduce compliance resulting in increases in both pressures (Table 4). Distinguishing between increases in peak airway pressure versus increases in both airway pressures can provide important information when troubleshooting decompensations of mechanically ventilated patients.

Ventilator alarm systems can provide an early warning of patient deterioration or of ventilator malfunction and should not be deactivated. The parameters monitored by the alarm systems vary between ventilators, but most modern systems provide notification of high or low V_E, low V_T, high or low respiratory rate, high peak airway pressure, or drops in PEEP or in the percentage of delivered O_2.

Complications of Mechanical Ventilation

Mechanical ventilation is occasionally associated with several complications which can be life-threatening (Table 5). Some complications are not directly related to mechanical ventilation, but to the ET or tracheostomy tubes used to provide positive pressure ventilation. Injury to the oropharynx or glottis can occur during intubation and the incidence of this complication correlates with the skill of the clinician performing the intubation.[5] Tracheal injury due to ET or tracheostomy tube cuffs is less common with the high compliance cuff designs in current use, but can still occur. Cuff pressures should be measured several times daily and maintained at less than 25 cm H_2O to lessen the probability of tracheal injury.[5,6]

Several adverse physiologic effects may result from mechanical ventilation. Increased intrathoracic pressure reduces ventricular preload and increases pulmonary vascular resistance which may reduce cardiac output. Increases in intracranial pressure and impaired hepatic venous drainage can also occur due to increased intrathoracic pressure. Reductions in cardiac output lead to corresponding reductions in renal function.

Fortunately, the effects of mechanical ventilation on renal and hepatic function are usually not clinically apparent.

Table 5. Complications of Mechanical Ventilation
Injuries by endotrachael or tracheostomy tube
Reduced cardiac output
Reduced hepatic function
Reduced renal function
Barotrauma
Nosocomial pneumonia
Ventilator malfunction

No well-conducted trial has ever established the superiority of any one weaning technique.

Fortunately, the effects of mechanical ventilation on renal and hepatic function are usually not clinically apparent.

Positive pressure ventilation may cause barotrauma in the form of interstitial emphysema, pneumomediastinum, subcutaneous emphysema, pneumothorax, and pneumoperitoneum. The incidence of barotrauma correlates with elevations in airway pressure, with most episodes occurring when peak airway pressures exceed 60 cm H_2O.[5]

Patients with high minute ventilations or obstructive lung disease may have insufficient time for complete exhalation with resultant air trapping and "auto" (intrinsic) PEEP. Auto PEEP can reduce cardiac output, increase the work of breathing, and may increase the incidence of barotrauma.[7]

Nosocomial pneumonia is common in mechanically ventilated patients, but has become less frequent with improved sterilization of respiratory therapy equipment and frequent changes in ventilator circuitry. Most cases of ventilator-associated pneumonia are probably related to impaired host defense mechanisms and to the inevitable colonization of the oropharynx and respiratory tract of intubated patients with nosocomial pathogens.

A last area of complication involves malfunction of the ventilator itself including the gas humidification system. Severe lung injury results from prolonged ventilation with non-humidified gas, making frequent checks mandatory to ensure the humidification system is functional.

Table 6. Methods of Weaning from Mechanical Ventilation	
Weaning Strategy	**Specific Weaning Technique**
Spontaneous breathing trials	Trials via T-piece circuit Trials via ventilator circuit
Partial ventilatory support modes	IMV/SIMV Pressure support ventilation

Weaning from Mechanical Ventilation

Weaning can be defined as the transition from full ventilatory support to unassisted ventilation. Weaning is not necessary in all situations. Patients with normal cardiorespiratory function awakening from surgery can usually be abruptly removed from the ventilator without untoward results. However, critically ill patients requiring mechanical ventilation generally need a more gradual reduction in ventilatory support. These patients are initially provided full ventilatory support, e.g., assist control mode, and weaning is initiated once improvement has occurred. The rapidity of weaning depends on the nature and severity of the acute illness and on the patient's underlying state of health.

Weaning Techniques

Although the proponents of various weaning techniques dogmatically tout the advantages of their method compared with others, no well-conducted trial has ever established the superiority of any one weaning technique.[4,5,8] Furthermore, the incorrect implementation of *any* weaning method can lead to respiratory muscle fatigue delaying extubation. All weaning techniques can be grouped according to two basic strategies (Table 6). The first strategy utilizes trials of spontaneous breathing of progressively longer duration separated by periods of "rest" on assist control mode (Fig. 1A). The original method is referred to as a "T-piece" or "flow-by" trial because the patient is removed from the ventilator circuit and placed on a separate tubing circuit via a T-shaped adapter that attaches to the endotracheal tube. This low resistance system minimizes the patient's work of breathing, but the lack of alarm systems necessitates intensive monitoring of the patient for respiratory distress throughout the trial. Spontaneous breathing trials can also be provided through the ventilator circuits of modern ventilators, but increased resistive loads are imposed on the patient. Trials via the ventilator circuit are advantageous in that the ventilator's alarm systems can provide notification of patient distress, and that most of these ventilators incorporate an "apnea mode" which initiates controlled mechanical ventilation if apnea occurs. Initial spontaneous breathing trials are brief while the patient is observed for signs of distress. Well-tolerated trials are continued for up to 30 min-

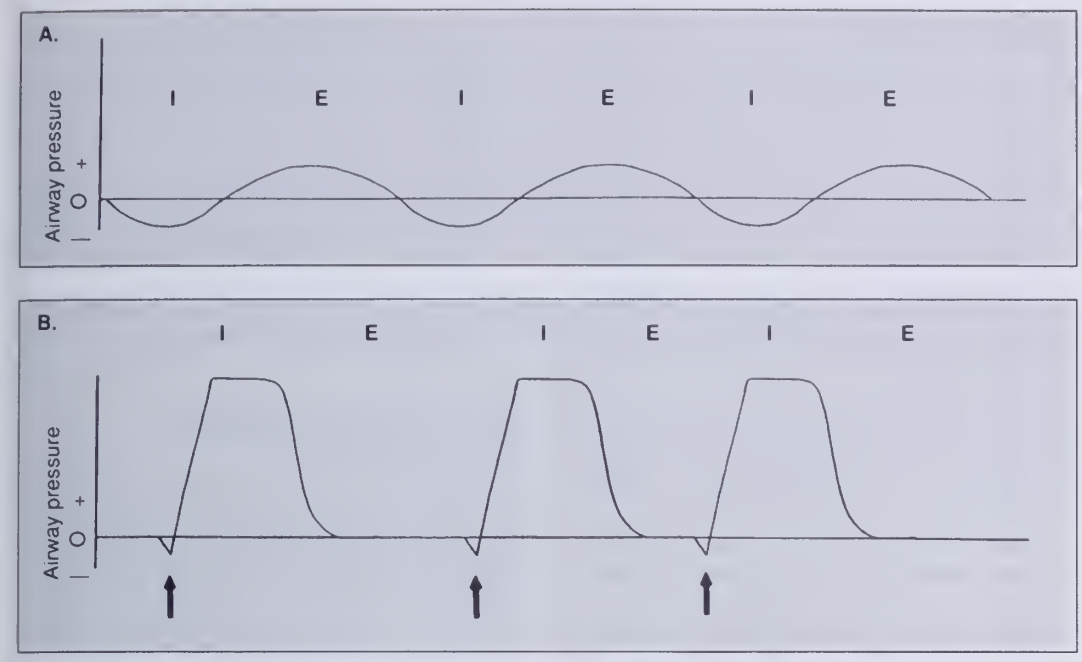

Figure 1. Figure 1A depicts the airway pressure versus time waveforms during spontaneous unassisted breathing. Airway pressure drops below baseline atmospheric pressure during inspiration (I) and increases above baseline during expiration (E). Figure 1B depicts the airway pressure versus time waveforms during pressure support ventilation. Each breath is patient-triggered when airway pressure drops below the baseline (see arrows). The ventilator assists each breath during inspiration (I) at the designated level of positive pressure until the patient's inspiratory flow lessens. No positive pressure is applied during expiration (E).

utes. ABGs are obtained prior to resuming assist control to monitor for the development of hypercapnia. The patient is placed in a sitting or semi-upright position and the percentage of inspired O_2 is increased by 10% to reduce the incidence of hypoxemia during the trial. Specific parameters for the early termination of the trial should be established, e.g. respiratory rate >40, pulse >120, oxygen saturation <90%, use of accessory muscles. Trials should be separated by at least one hour of rest[9] and suspended at night to allow sleep. If trials are provided via the ventilator circuit, then 5-10 cm of pressure support ventilation should be added to compensate for the increased work of breathing imposed by the ventilator circuity.[5,10] Extubation should be considered when trials of several hours duration are tolerated without distress or deterioration in ABGs.

The second weaning strategy uses partial ventilatory support modes in the transition from full ventilatory support to unassisted ventilation. Intermittent mandatory ventilation (IMV) is a form of partial ventilatory support long used for weaning. IMV allows the patient to take spontaneous breaths between machine-delivered breaths. Weaning occurs by reducing the rate of machine-delivered breaths until the patient assumes the full burden of ventilation. Rate reductions are usually made in increments of 2 breaths/minute. ABGs are measured thirty minutes after each reduction and the patient is observed for distress. However, IMV has been replaced by synchronized IMV (SIMV) on most new ventilators and SIMV imposes significant increases in the patient's work of breathing.[5] Low levels of pressure support (5-10 cm H_2O) help compensate for the resistive loads placed upon the patient by SIMV.[5] In the author's opinion, SIMV should not be used without concomitant pressure support ventilation. Patients requiring prolonged weaning may develop respiratory muscle fatigue on low rates of IMV or SIMV and should be "rested" at night on assist control mode or a high rate of IMV.[12]

A second technique of partial ventilatory support is pressure support ventilation. In addition to its uses to reduce the work of breathing during SIMV and spontaneous breathing trials, pressure support alone is used as a weaning technique. During pressure support ventilation each breath is patient-initiated but then boosted at the designated pressure level until the patient's inspiratory flow declines at the end of inspiration (Fig. 1B). No minimum respiratory rate or tidal volume is guaranteed using pressure support venti-

Clinicians often are confused by the term continuous positive airway pressure (CPAP).

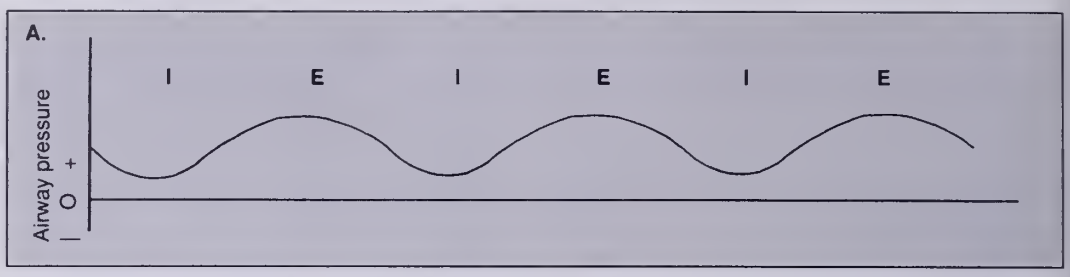

Figure 2. Airway pressure versus time waveforms during continuous positive airway pressure. The waveform resembles that of spontaneous breathing (Figure 1A) except that airway pressure remains positive throughout the respiratory cycle. Fluctuations in pressure occur during inspiration (I) and expiration (E).

Patients under consideration for extubation should be hemodynamically stable and able to protect their airway.

lation. Pressure support is a comfortable mode as the patient controls rate, tidal volume, inspiratory flow rate, and the inspiratory:expiratory ratio. When used for weaning, the level of pressure support is initially titrated to maintain the same minute ventilation that was provided on full ventilatory support. The pressure support level is then reduced in 3-5 cm H_2O increments with ABGs and observation for distress following each reduction.[12] Most patients are extubated from a pressure support level of 5 cm H_2O. Patients requiring several days for weaning should receive full ventilatory support at night to allow sleep and avoid respiratory muscle fatigue.

Use of CPAP During Weaning

Clinicians often are confused by the term *continuous positive airway pressure* (CPAP). CPAP systems maintain positive airway pressure throughout the respiratory cycle, although pressures drop slightly during inspiration (Fig. 2). Unlike pressure support, the patient receives *no* increase in pressure during inspiration to augment ventilatory efforts. PEEP as applied during assist control mode is termed CPAP when the patient is changed to IMV or a spontaneous breathing mode. The routine use of low level CPAP during weaning is controversial. In diseases like ARDS that reduce lung volume, CPAP during weaning increases lung volume preventing hypoxemia from microatalectasis and also may reduce the work of breathing.[12]

CPAP is provided to patients spontaneously breathing via a T-piece circuit by adding a CPAP valve to the end of the circuit. CPAP can be applied by modern ventilators to patients on SIMV or spontaneously breathing through the ventilator circuit. CPAP provided through the ventilator circuit imposes resistive loads on the patient and low level pressure support should be added to avoid *increasing* the patient's work of breathing.

Determining Suitability for Extubation

Patients under consideration for extubation should be hemodynamically stable and able to protect their airway. Comatose patients and those with significant glottic dysfunction require tracheotomy to provide some protection from aspiration and to facilitate suctioning. Oxygenation should be adequate as defined by a PaO_2 greater than 60 mm Hg on 35% oxygen and no more than 5 cm H_2O of PEEP/CPAP.[5] Brief trials of spontaneous breathing, e.g. 30 minutes, via a T-piece circuit or on pressure support of 5 cm H_2O should be tolerated without the development of acute respiratory acidosis (pH <7.30),[13] marked use of accessory muscles of respiration, or other signs of respiratory distress. A number of "weaning parameters" have been proposed to estimate the ventilatory capability of the patient (Table 7). These include a maximum inspiratory pressure less than -30 cm H_2O, a vital capacity of at least 10 ml/kg, a resting V_E less than 10 l, and a maximal voluntary ventilation that is twice the V_E.[5,12] However, prospective studies of these parameters have demonstrated them to have a poor predictive value in identifying weaning success in some groups of patients.[14,15]

Other studies have noted that patients failing to wean develop a pattern of rapid shallow breathing during unassisted respiration.[16] This lead to the development of the rapid shallow breathing index, expressed as the ratio of respiratory rate (f) to tidal volume (VT) in liters. Measurements of f and V_E are obtained after removing the patient from the ventilator and allowing one minute of spontaneous breathing. VT is calculated by dividing the V_E by the f. A rapid shallow breathing index (f/VT ratio) less than 100 predicts successful weaning. The index is easily determined at the bedside and more accurately predicts successful weaning than do the traditional "weaning parameters."[14]

Causes of Weaning Difficulty

Hemodynamic instability, persistent hypoxemia, and psychologic factors can complicate the weaning process. However, ventilatory insufficiency is the most common factor delaying extubation. A minority of patients have ventilatory insufficiency due to reductions in respiratory drive from sedating medications or neurologic insults. More commonly, respiratory drive is intact but ventilatory capability can not match ventilatory demands. CO_2 production may be increased by fever, by feeding excessive amounts of carbohydrates, or by an increased work of breathing from persistent pulmonary disease. Persistent disease also may increase physiologic dead space, decreasing the lungs' efficiency in eliminating CO_2. Numerous factors may weaken the respiratory musculature reducing ventilatory capacity. Malnutrition, hypocalcemia, hypophosphatemia, hypokalemia, and hypothyroidism are treatable causes of respiratory muscle weakness. Metabolic acidosis and metabolic alkalosis are acid-base disturbances which can hamper weaning. Respiratory muscle fatigue may result from poor weaning technique, the use of SIMV or CPAP on older generation ventilators, or from a high resistive load imposed by a small endotracheal tube diameter (<7.5 mm).

Timing of Tracheotomy

The timing of tracheotomy has been a subject of ongoing controversy. No convincing data exist that translaryngeal intubations of several weeks duration result in a higher incidence of significant upper airway complications in comparison to tracheotomy.[17] Furthermore, no prospective studies have confirmed that tracheotomy facilitates weaning.[17] For awake patients tracheotomy offers the advantages of increased comfort, easier mobilization from bed, and the opportunity to resume eating. Several authorities recommend tracheotomy for patients not expected to be weaned after 14 days of translaryngeal intubation.[12,17] However, some centers maintain translaryngeal intubations beyond 21 days unless complications like sinusitis or nasal/labial erosions develop.

Extubation

Extubation is performed when the weaning parameters, ABGs, and the bedside examination during unassisted ventilation all suggest a favorable outcome. Patients are generally extubated early in the day, maximizing the availability of physicians and staff should reintubation prove necessary. The patient is placed in an upright position and the airways and oropharynx are thoroughly suctioned before extubation. Following extubation the percentage of inspired O_2 is usually increased by 10% and provided by humidified mask. The patient should be examined for the presence of stridor and signs of respiratory distress. ABGs are obtained 30 minutes after extubation and are generally repeated a few hours later. Up to 20% of patients require early reintubation making careful patient monitoring essential for several days following extubation.[5]

Table 7. Parameters for Predicting Weaning Success

Oxygenation	PaO_2 > 60 on 35% O_2 and PEEP≤ 5	
Ventilation	Vital capacity	> 10 ml/kg
	Max. inspiratory pressure	≤ — 30 cm H_2O
	Resting $\dot{V}_E$	≤ 10 1/min
	Max. voluntary ventilation	≥ 2 × resting $\dot{V}_E$
	Rapid shallow breathing index	$\left(\frac{f}{VT\ ratio}\right)$ <100

Summary

Mechanical ventilation can provide life saving support to critically ill patients with respiratory failure. The implementation of appropriate monitoring techniques and an awareness of potential complications can increase the safety of mechanical ventilation. Several efficacious weaning methods can be utilized and none is clearly superior to the others. The improper use of any weaning technique can result in respiratory muscle fatigue delaying extubation. Determining patient suitability for extubation is an inexact science. The physical examination during a spontaneous breathing trial and the use of the rapid shallow breathing index can assist the clinician in the difficult decision as to the timing of extubation.

Acknowledgment

The author is indebted to Ms. Jan Gamble for typing the manuscript and to Gary Kinasewitz, MD, for helpful comments during its preparation.

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The Author

Brent Reiff Brown, MD, is an assistant professor of medicine at the University of Oklahoma College of Medicine in Oklahoma City.

Vaginal Hysterectomy: The Use of a Self-Retaining Retractor

Saeed Mahmoodian, MD

This report concerns the indications, technique, and safety of vaginal hysterectomy among 213 women. It further examines the comparative risks of complications and costs between 107 patients whose operations were performed with the use of the new retractor during the 5-year period ended June 1991 and 106 other patients whose surgeries were done during 4 preceding years when the retractor was not available. The selected characteristics of the patients undergoing vaginal hysterectomy in the two groups were largely similar. When the retractor was used, suturing the cuff and pedicles was easier, as the retractor facilitated good exposure and better visibility. In no case was there any injury or the need for transfusion. Of 7 patients with morbidity, 1 had no demonstrable cause, 3 had urinary tract infection, and 3 had cuff cellulitis of which one progressed into abscess requiring transvaginal drainage 6 days post-operation. My experience with 107 vaginal hysterectomies using the self-retaining retractor demonstrates that the retractor was instrumental in significantly reducing operative time, blood loss, and morbidity.

From the Department of Obstetrics and Gynecology, United Hospital Center, Inc., Clarksburg, West Virginia.Read before the American College of Obstetricians and Gynecologists, District IV Annual District Meeting, San Juan, Puerto Rico, November 14-19, 1992.

Direct correspondence to Saeed Mahmoodian, MD, 3703 Meadowbrook Drive, Rapid City, SD 57702.

WHEN I was chief resident some 23 years ago, our department lost a brilliant resident who simply could not go through holding retractors for two hours during vaginal surgery after a sleepless night delivering babies. Discerning in private practice assistants a mute discontent in retracting the vaginal wall, I thought there must be a better way. Wishing to reduce the hardship and increase the safety, I developed a self-retaining retractor which was awarded a United States patent for 17 years beginning May 15, 1990. This vaginal retractor comprises a pair of parallel supporting bars arranged in a frame so as to move toward and away from each other by a pair of turnbuckle mechanisms that keep the vaginal walls separated, allowing one surgeon to perform surgery alone (Fig. 1).

Even though hysterectomy rates have been declining, it is still the most frequently performed major operation in the United States.[1] There is, however, a significant difference in hysterectomy rates among communities within a single state[2] as well as among regions within the United States.[1] These disparities are more prominent when they relate to vaginal hysterectomy, not because hysterectomy may exert a unique emotional, medical, sexual, and economic effect on women but because of the differences in the training and experience of the physician and the style of medical practice. In a 3-year prospective multicenter, observational study coordinated by the Centers for Disease Control, Dicker et al[3] found the overall rate for abdominal hysterectomy was twice that for the vaginal approach, 69.3% to

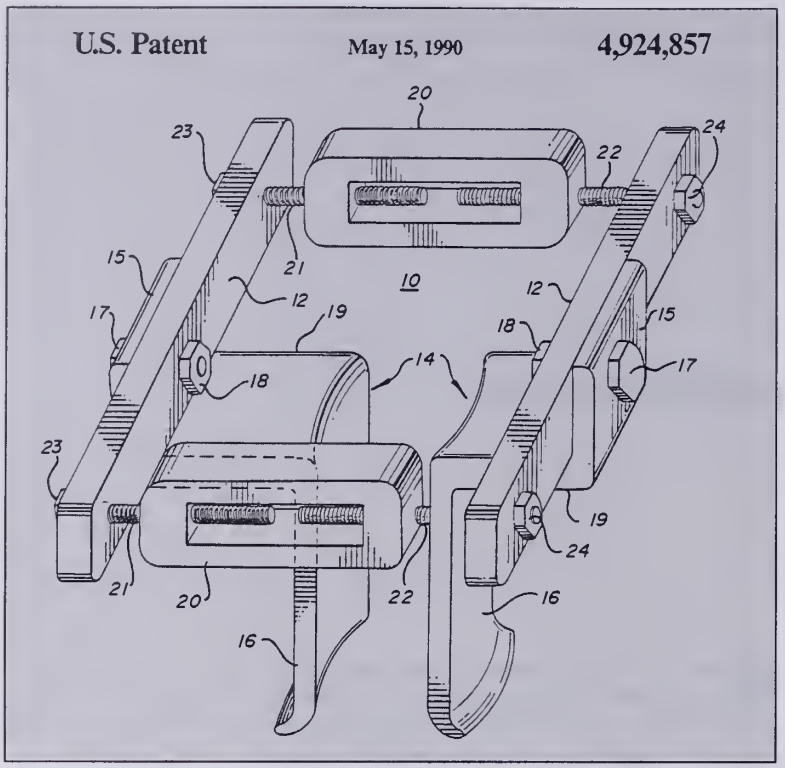

Figure 1. Diagram of vaginal retractor as it is registered in the Office of the United States Patents and Trademarks.

Table 1. Selected Characteristics of 213 Patients Undergoing Vaginal Hysterectomy

Characteristic (%)		Vaginal Hysterectomy: With Retractor 107 Cases	Without Rectactor 106 Cases	Total 213 Cases
Age	21-25	3.8	1.9	2.8
	26-44	85.0	78.3	81.7
	45-73	11.2	19.8	15.5
Parity	1	12.2	4.7	8.5
	2-4	77.5	75.5	76.5
	5-8	10.3	19.8	15.0
Previous tubal ligation		29.9	36.4	33.3
Previous Cesarean section		5.6	5.7	5.6
Previous other pelvic surgeries		18.7	13.2	16.0
Obesity*		44.0	37.7	41.0

*Defined as weight more than 20% above the ideal body weight.

30.7%. In recent years vaginal hysterectomy has gained in the U.S. a considerable popularity.[4] The reasons, as are shown by this prospective study, are (1) low morbidity as well as comparatively mild postoperative discomfort due to less extent of the peritoneal incision, and (2) vaginal route provides an excellent opportunity for the correction of frequently coexistent uterine vaginal descent and pelvic relaxation.

Materials and Methods

Between July 1986 and June 1991, the author, without an assistant surgeon, performed 107 vaginal hysterectomies using the new self-retaining retractor. For comparison in this study, 106 other patients also are included whose operations were performed during four preceding years when the retractor was not available.

Patients' characteristics (Table 1), age, parity, history of previous surgery, and weight were similar; obesity was encountered often. The retractor was of special help in this group of patients as it is easily adjustable to provide a wide, clear operating area. The ages of the patients ranged from 21 to 73 years, with a mean age of 38 years. Parity ranged from 1 to 8; 87% were multiparas. All patients had Papanicolaou smears and colposcopy, with colposcopically directed biopsies when indicated.

Because of quality assurance requirements, the necessity of some hysterectomies during the period of this study was under intense scrutiny. Therefore, only patients with significant indications were selected to warrant the procedure (Table 2). The size of the uterus was considered an important factor in evaluating the feasibility for vaginal hysterectomy; however, the mobility and accessibility of the uterus was the single most determining factor in the suitability for transvaginal approach. All patients received preoperative antibiotics.

The operative technique for 213 vaginal hysterectomies was constant. It was essentially the method described in a standard vaginal surgery textbook.[5]

Results

The most common preoperative indication for vaginal hysterectomy was pelvic relaxation while cervical dysplasia (CIN II, III) constituted the second most frequent (Table 2). Overall, 33.3% and 5.6% of 213 women undergoing vaginal hysterectomy in this study had had previous tubal sterilization or Cesarean section, respectively.

The mean surgical time for hysterectomy alone was 48 minutes; it ranged from 28 to 82

minutes. When the retractor was used, there was an eight-minute average time saving. Blood loss, estimated from 75 to 600 cc, averaged 200 ml. No patient received blood transfusion. Similarly, in this group of patients, no one required an intraoperative surgical procedure to repair bladder or bowel trauma or to control bleeding and/or to complete hysterectomy. The complications are summarized in Table 3. There were no cases of death, postoperative ileus, thrombophlebitis, bowel injuries, urethral injuries, pneumonia, or atelectasis.

Fourteen patients (eleven in the group using the retractor) underwent simultaneously incidental (5) or therapeutic (9) oophorectomies. Three of the therapeutic oophorectomies were done as prophylaxis on patients with family history of ovarian cancer. The retractor made removal of the ovaries easily or readily available (Fig. 2). The extra surgical time spent on each of these patients averaged 9.8 minutes. All of the women undergoing vaginal hysterectomy with principal preoperative diagnosis of pelvic relaxation underwent concurrent anterior and posterior colporrhaphy.

The average length of hospital stay for the 213 patients undergoing vaginal hysterectomy was 3.7 days. The length of hospital stay progressively declined, averaging 2.8 days for those whose operations were done during the period of 1986 to 1991. No patient was readmitted to the hospital. No late sequelae of hysterectomy such as depression, sexual dysfunction, "residual adnexa syndrome," or fallopian tube or vaginal vault prolapse were experienced among 190 patients who were not lost to follow-up 1 to 10 years postoperation.

Discussion

An indicated hysterectomy is one of the few operations that help significantly to improve the quality of life of women. However, the inherent risks coupled with the associated complication[3,6] and sizeable proportion of malpractice verdicts[7] warrant critical consideration of indications and choice of surgical approach.

Whenever possible, the vaginal route should be preferred because of low complication rate, low mortality rate, and low rate of postoperative morbidity. Breen and Scutella[8] report less than one death per 1,000 vaginal hysterectomies, well below the average mortality rate associated with abdominal hysterectomy.[9] From a collective review of 568 cases of vaginal and 1,283 cases of abdominal hysterectomy Dicker et al[3] reported an overall complication rate of 42.8% and 24.8% for abdominal and vaginal hysterectomies, respectively. These authors, as well as others,[6,10-12] attribute the lower frequency of febrile and infectious morbidity to the use of prophylactic antibiotics in vaginal hysterectomy.

In this study is reported one of the lowest complication and postoperative infection rates (Table 3). This low rate of complication is credited to the use of preoperative antibiotics, opened cuff technique, and the procedure of attaching the uterosacral and cardinal ligaments to the corners of the vaginal membranes in all of the patients. It is believed[6,13] this procedure helps prevent vault prolapse and postoperative hemorrhage as it ties the angles of the vagina, the common site of postoperative bleeding. The self-retaining retractor made suturing the cuff and ligaments to the vaginal angles easier, as it facilitated good exposure and better visibility (Fig. 3).

The advantages of vaginal hysterectomy are easily understood: less blood loss, reduced anesthesia time, and less handling of the bowels which reduces the disruption of bowel functions and the risks of postoperative peritonitis and paralytic ileus. Vaginal hysterectomy, furthermore, avoids the discomfort and risk of infection associated with an abdominal incision.[4]

The self-retaining retractor made suturing the cuff and ligaments to the vaginal angles easier, as it facilitated good exposure and better visibility.

Table 2. Principal Preoperative Indications Among 213 Women Undergoing Vaginal Hysterectomy

Indication	No.	%
Abnormal uterine bleeding	18	8.5
Adenomyosis	5	2.3
Cervical dysplasia (CIN II, III)	35	16.4
Chronic pelvic pain	32	15.0
Leiomyomata	2	0.9
Pelvic relaxation	121	56.8

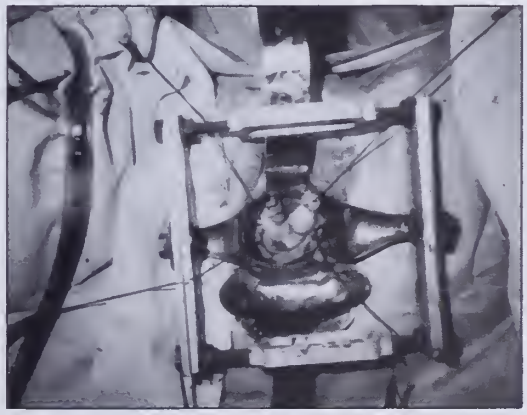

Figure 2. Ovaries are exposed and are about to be removed.

Table 3. Comparison of Complications Among 213 Patients Undergoing Vaginal Hysterectomy

Complication (%)		Vaginal Hysterectomy: With Retractor 107 Cases	Without Rectactor 106 Cases	Total 213 Cases
Febrile morbidity	Source unidentified	0.9	0.0	0.4
	Urinary tract infection	2.8	2.8	2.8
	Vaginal cuff infection	2.8	4.7	3.7
Hemorrhage requiring transfusion	Intraoperative	0.0	0.9	0.4
	Postoperative	0.0	1.8	0.9
Unintended surgical procedure	To complete hysterectomy	0.0	0.9	0.4
	To control bleeding	0.0	1.8	0.9
	To repair bladder trauma	0.0	0.9	0.4

The prophylactic removal of ovaries at the time of hysterectomy has been the subject of discourse and writing for several decades.

In comparison, patients who undergo vaginal hysterectomy have shorter hospital stays and recuperation periods than those who have an abdominal hysterectomy.[3,14] This study demonstrates that vaginal hysterectomy with early discharge can be compared with laparoscopy-assisted vaginal hysterectomy or laparoscopic supracervical hysterectomy in terms of safety, cost, and patient acceptance. It may prove superior in terms of avoiding three stab wounds and prolonged anesthesia, anxiety of occasional continued postoperative menstruation, and lifetime surveillance for cervical intraepithelial neoplasia and complications unique to laparoscopy and faulty Verres needle placement and pneumoperitoneum induction.[15]

Hemorrhage is a frequent complication of vaginal hysterectomy. However, changing indications and improved surgical techniques have significantly reduced this complication as the rate of blood loss requiring transfusion has steadily decreased from 13 per 100 operations reported by Ledger and Child[17] in 1973, to 8 per 100 reported by Dicker et al[3] 1982, to the rate of 1.4% in the present series. The rate of this complication can be corrected to 2.8% and 0%, respectively, for 106 patients undergoing vaginal hysterectomy in the period between 1982 to 1986 and 107 whose operations were done during the 5-year period ending June 1991.

Figure 3. Vaginal cuff is pursestringed; peritoneal cavity is ready to be closed.

The prophylactic removal of ovaries at the time of hysterectomy has been the subject of discourse and writing for several decades. The death of Gilda Radner, the well-known comedienne, in 1989 has provoked renewed interest. Studd[18] recommends prophylactic oophorectomy at abdominal hysterectomy in all women over the age of 40 years. Considering that 1 in 70 women will develop ovarian cancer in her lifetime[19] and that both the present study, albeit statistically small, and the study of Capen and his colleagues[20] demonstrate that the ovaries could be removed without much difficulty at the time of vaginal hysterectomy, without significantly increasing morbidity, the author recommends applying the same logic at the time of vaginal hysterectomy. The retractor may prove a special help in such a procedure, as its smooth blades open the vagina wide without obstruction while still providing convenient adjustment.

In conclusion, this report may establish sufficient information to permit critical evaluation of the new retractor which was originally designed to enable the vaginal surgeon to perform vaginal hysterectomy without the need for an assistant.

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The Author

Saeed Mahmoodian, MD, currently is chairman of the Department of Obstetrics and Gynecology at the PHS Hospital, Pine Ridge, South Dakota.

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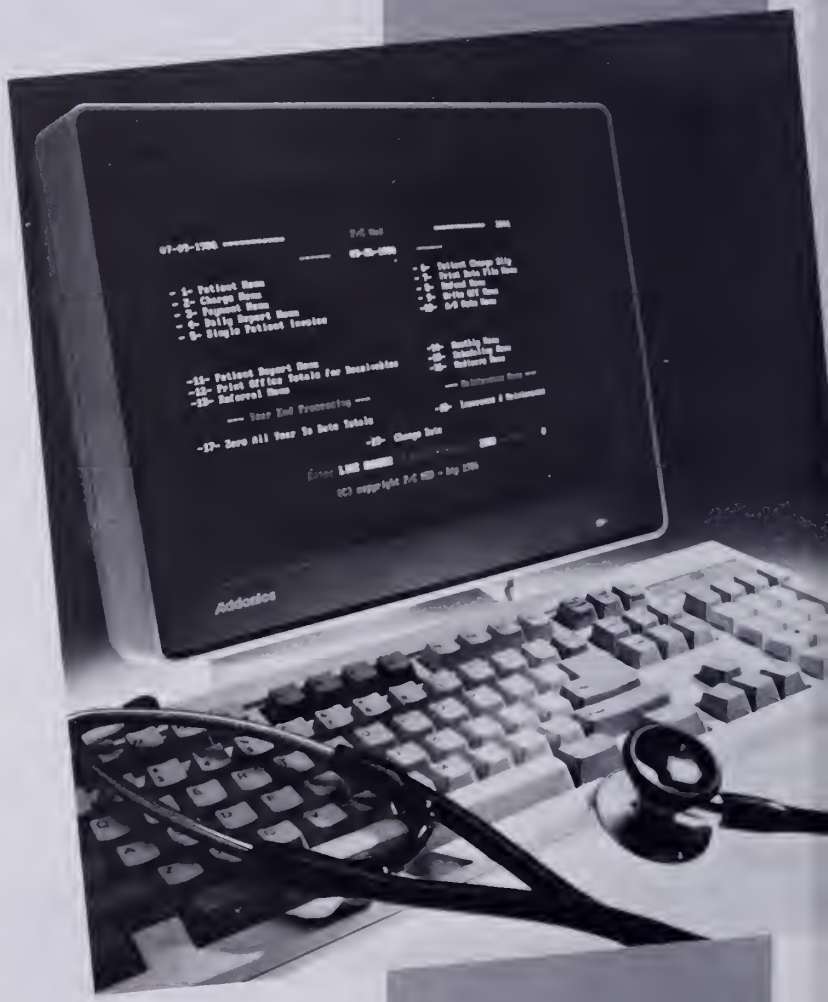

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Women physicians honored this month by national, local groups

September is Women in Medicine Month, the time designated by the American Medical Association to create heightened awareness of and recognition for women physicians and to increase their participation in organized medicine. The AMA is encouraging all medical societies to develop on-going membership recruitment and retention programs.

The OSMA's Women in Medicine Committee is a prime example of such a program. It approaches women physicians as physicians and students first and foremost, while recognizing that they have concerns and needs unique to their gender.

And it appears that the committee's efforts have been effective. In 1993 Oklahoma led the nation in its percentage of female medical doctors in organized medicine—61.39% of the 1,093 women physicians in the state belonged to the AMA—and 20.95% of all Oklahoma medical doctors were women. (Oklahoma also had the nation's highest percentage of medical doctor in organized medicine—77.91% of the state's 5,217 MDs were AMA members.)

Nationwide, women now represent 19% of all physicians, compared to just 7.6% in 1970, and projections are that by the year 2010, women will constitute 30% of all U.S. physicians. The number more than doubled between 1970 and 1980, and by 1990 had increased more than 300%. There were more than four times as many women physicians in 1990 as in 1970—125,782 vs. 25,401.

In 1969-70, 9.4% of all medical school applicants were women; by 1992-93 the percentage had increased to 41.8%. Total enrollment of women during the same period grew from 9.0% to 39.0% and exceeded 50% at nine schools.

It's your association

When it comes to leadership, it's what's up front that counts

Presumably most OSMA members know the names of their elected leaders, but perhaps less well known is the actual election process and the specific duties assumed by those officers after their election.

Their election, terms of office, and duties are explained below in chapters from the OSMA bylaws (as revised May 1993).

Current officers of the association are President Jay A. Gregory, MD, Muskogee; President-Elect Larry L. Long, MD, Oklahoma City; Vice-President David L. Harper, MD, Tulsa; Secretary-Treasurer Carol Blackwell-Imes, MD, Oklahoma City; Speaker of the House Mary Anne McCaffree, MD, Oklahoma City; Vice-Speaker Boyd O. Whitlock, MD, Tulsa; Chairman of the Board Douglas C. Hubner, MD, Tulsa; and Vice-Chair Chester L. Bynum, MD, Norman.

CHAPTER VI
ELECTION OF OFFICERS, TRUSTEES

Section 1.00 Officers and Trustees. The general officers of the Association shall be the President, President-Elect, Immediate Past-President, Vice-President, Secretary-Treasurer, Speaker of the House of Dele-

Bylaws *(continued)*

gates, Vice-Speaker of the House of Delegates, Chair and Vice-Chair of the Board of Trustees. The composition of the Board of Trustees is defined in Chapter V, Section 1.00 of these Bylaws.

Section 2.00 Electorate. The House of Delegates shall serve as the electors of the general officers, except the offices of Chair and Vice-Chair of the Board of Trustees, and the Trustees and Alternate Trustees of the Association at the annual meeting.

Section 3.00 Tenure. The President-Elect and the Vice-President shall be elected for a term of one year; the Secretary-Treasurer, Speaker and Vice-Speaker of the House of Delegates shall be elected for terms of two years each. At the expiration of his/her term, the President-Elect shall become President for a period of one year. At the end of that year, he/she shall then serve one year as Immediate Past-President. Trustees and Alternate Trustees shall be elected for concurrent terms of three years each, provided that the tenure of a trustee is limited to two consecutive full terms.

Section 4.00 Eligibility. Any Active member in good standing, who has full membership rights and privileges except as otherwise specified in these bylaws, may be elected as a general officer, Trustee or Alternate Trustee.

Section 4.01 Multiple Offices. No member may hold more than one general office at a time. A general officer may also be a Delegate or Alternate Delegate to the American Medical Association, or a member of the plico Board of Directors, but not both, with the exception of the President.

Section 5.00 Nominations. Candidates for general officer positions, except the offices of Chair and Vice-Chair of the Board, may be nominated by any member of the House of Delegates. Candidates for Trustee or Alternate Trustee must be nominated by a Delegate from their Trustee Districts, as provided in Chapter V, Sections 1.00 and 2.00 of these bylaws.

Section 6.00 Time. Nominations for all offices elected by the House of Delegates shall be made only in the opening session of its annual meeting.

Section 7.00 Election. Elections shall be conducted before the close of the annual meeting of the House of Delegates.

Section 8.00 Ballot. All contested elections shall be by ballot, and a majority of the votes cast shall be necessary to elect. If no nominee receives a majority of the votes cast on the first ballot, the nominee receiving the fewest votes shall be dropped from the list and a new ballot taken. This procedure shall be continued until one of the nominees receives a majority of the votes cast. Where there is only one nominee for an office, a majority vote, without ballot, shall elect. In the event of a tie between the two nominees receiving the least number of votes, a new ballot shall be taken.

Section 9.00. General Officer Vacancies. Vacancies in any general office, except President, President-Elect, Chair or Vice-Chair of the Board, and Speaker of the House of Delegates, shall be filled by appointment of the President, with the approval of the Board of Trustees, effective until the next annual meeting, at which time the House of Delegates shall elect a successor to either begin a new term or to complete the unexpired term. A vacancy in the office of President shall be filled automatically by the President-Elect. A vacancy in the office of Chair of the Board shall be filled automatically by the Vice-Chair, and a new Vice-Chair shall be elected by the Board. A vacancy in the office of Speaker of the House of Delegates shall be filled automatically by the Vice-Speaker. If the office of President-Elect becomes vacant, the President shall immediately call a special session of the House of Delegates for the purpose of filling the vacancy by election.

Section 10.00 Trustee Vacancies. A vacancy occurring in the position of District Trustee shall be filled automatically by the authorized Alternate Trustee. A vacancy occurring in the position of District Alternate Trustee shall be filled by appointment of the Board of Trustees, after considering nominations received from the presidents of affected component medical societies. Such appointments by the Board of Trustees shall expire at the next annual meeting, at which time the House of Delegates shall elect a successor to fill the unexpired term.

Chapter VII
Duties of Officers

Section 1.00 President. The President shall be a voting member of the House of Delegates, Board of Trustees, Council on Planning and Development, and of certain committees as provided in these bylaws, and shall be an ex-officio member of all other appointed councils and committees. The President shall be the chief representative of the Association and shall coordinate and supervise all Association activities.

Section 2.00 President-Elect. The President-Elect shall become President upon installation at the annual meeting following his election. While in office the President-Elect shall be a member of the House of Delegates, the Board of Trustees, the Council on Planning and Development and of certain committees as provided in these bylaws. In the event of the death, resignation, removal from the state, disqualification or disability of the President, as determined by the Board of Trustees, the President-Elect shall succeed to the Presidency for the unexpired term.

Section 3.00 Vice-President. The Vice-President shall assist the President in the coordination and supervision of all Association activities. The Vice-President shall be a member of the House of Delegates, Board of Trustees, and the Council on Planning and Development. In the event of vacancy in the offices of President and President-Elect, the Vice-President shall become acting President for the unexpired term.

Section 4.00 Secretary-Treasurer. The Secretary-Treasurer shall be a member of the House of Delegates, the Board of Trustees, and the Council on Planning and Development and shall keep the minutes of all meetings of the House of Delegates. The Treasurer shall give bond in the amount specified by the Board of Trustees and shall be custodian of all funds of the Association and shall make all authorized disbursements on voucher, signed by the Chief Executive Officer. The Treasurer shall render an annual account of all receipts, expenditures, funds invested and on hand to the Board of Trustees and House of Delegates, and shall open the books and records to such auditor inspection as may be ordered by the Board of Trustees or House of Delegates. Reserve funds shall be invested on order of the Board of Trustees. When authorized by the Board of Trustees, money may be borrowed in the name of the Association and property of the Association pledged as security for the repayment thereof.

Section 5.00 Speaker of the House of Delegates. The Speaker shall be a member of the House of Delegates, the Board of Trustees, and of committees as provided in these bylaws and shall preside at all meetings of the House. In the absence of the Vice-Speaker any member of the House may be designated by the Speaker to preside temporarily.

Section 6.00 Vice-Speaker of the House of Delegates. The Vice-Speaker shall be a member of the House of Delegates and the Board of Trustees and shall preside in the absence of the Speaker. The Vice-Speaker shall become Speaker for the unexpired term upon occurrence of a vacancy in that office.

Section 7.00 Immediate Past-President. The Immediate Past-President of the Association shall remain as a general officer of the Association for a period of one year following the presidential year and shall serve as Chair of the Council on Planning and Development, and as a member of the Board of Trustees and as a member of other committees as specified in these bylaws.

MENTAL ILLNESS AWARENESS WEEK
October 2-8, 1994

Our rate of drowning deaths is 25% above the national average

Submersions (drowning or near drowning), are the third leading cause of unintentional injury death in Oklahoma; the drowning death rate is about 25% higher than the national level. There are approximately 120 submersion injuries each year in Oklahoma, including 85 to 90 deaths. Since fatal and hospitalized submersion injury became a reportable condition statewide in Oklahoma in 1987, the occurrence, patterns, and causes of injury have been studied and efforts made to reduce the number and severity of injuries. Although submersions peak in the under-5 and 15-24-year age groups, persons of all ages are susceptible to water hazards. In addition to the known risks of pools, spas, bathtubs and other water deposits in homes, Oklahomans have a high exposure to water activities and hazards specific to natural bodies of water (ponds, lakes, rivers, streams) and flood waters. The state reportedly has the highest shoreline mileage in the nation and one of the highest water-to-land ratios; there are approximately 250,000 farm ponds totaling 515 square miles, 1,098 square miles of lakes, and 12,294 streams.

Among children under five years of age, 31% of submersion injuries are fatal. Fatal events are higher in ponds and natural bodies of water, likely due to time-to-discovery and consequent submersion time, and lack of immediate resuscitation. Pool submersions are usually noted quickly and resuscitation measures are more readily available. Sixty-three percent of submersions among Oklahoma children 5 to 14 years of age are fatal. These injuries most often occur in ponds and natural waters, and are commonly linked to risk-taking behavior (swimming and diving in unknown waters) and swimming alone.

About three-fourths of submersion injuries in Oklahomans 15 years of age and older occur in natural bodies of water while they are swimming, boating, fishing, or engaged in other water activities; some injuries are related to motor vehicle crashes and to flooding steams and rivers. Ninety percent of all submersion injuries in persons 15 and older are fatal, and chance of survival decreases with age. Alcohol use, swimming or fishing alone, and non-use of protection such as personal flotation devices (PFDs) contribute to submersion

(continued)

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Drowning deaths *(continued)*

injury and the high proportion of fatal events.

Lack of swimming ability, developmental factors, the presence of pre-existing conditions, reckless behavior, and environmental hazards contribute to submersion injury. Preventing access to water with childproof barriers, especially fencing, and close supervision are the main ways to reduce drowning in young children. Knowledge and use of water safety habits, such as never swimming alone, knowing the body of water before entering, abstaining from alcohol, use of PFDs when boating/fishing, and not overestimating swimming skills will help prevent submersion events in older children and adults. In addition, knowledge of appropriate electrical installation and device use in or near water is important. Quick retrieval of the victim and appropriate resuscitation are key in preventing hypoxemia and subsequent death or disability. Emergency department staff often use a submersion or near-submersion event as a time to educate and counsel families and communities about drowning hazards and safety measures. Physician support of water safety legislation, parental and community education, and CPR training is important in increasing awareness about submersion injury prevention. For more information about submersion injury, contact the Injury Prevention Service (405) 271-3430).

LETTERS

Guns and the AMA

To the Editor: I recently read your editorial in the JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION ["Who Has Guns?" July 1994] regarding Article II of the United States Constitution and would like to applaud you. I have been very much opposed to the stand on the issue of gun control recently taken by the American Medical Association, and if we were not forced to be in the AMA by reason of unified membership I would have resigned from the AMA over this issue. I appreciate the stand you have taken on this issue because I personally feel the current legislative attempts on gun control are a direct attempt at eroding constitutional rights and freedoms.

There are many people who enjoy the safe sporting use of firearms and many people who also use them in the defense of their loved ones and personal property. Law abiding people take the proper responsibility in the use of firearms, and gun control laws only take away our means of defense against the ever-increasing aggressiveness of criminals. It appalls me that many of my colleagues in the medical profession have such a short-sighted vision of things to sign on to a blatant attempt at doing away with the hard fought freedoms of our previous generations.

Once again, I applaud you for this editorial.

—James O. Shaw, MD
Altus

To the Editor: Thank you for your May editorial. More than a few of us share your views on gun control and the Second Amendment.

Please convey your thoughts to the AMA for us.

Again, thank you.

—Chris Olson, MD
Tulsa

DEATHS

Jess Duval Herrmann, MD
1907 - 1994

Jess D. Herrmann, MD, one of the JOURNAL's Leaders in Medicine (June 1990), died August 16, 1994, in Oklahoma City. Born in Denison, Tex., Dr. Herrmann was graduated from the University of Oklahoma School of Medicine in 1931. A neurosurgeon, he was clinical professor and head of neurosurgery at his alma mater and was named clinical professor emeritus in 1968. During World War II he served with the U.S. Army in the American and Pacific theaters from 1942 to 1946, attaining the rank of lieutenant colonel and earning a number of service awards, including the Bronze Star. Aside from his military service, Dr. Herrmann's years of practice were spent entirely in Oklahoma City. In 1956-57 he served as president of the American Academy of Neurological Surgery. He retired and moved to Arkansas in 1967, returning to Oklahoma City just a few years ago.

Mark Daniel Holcomb, MD
1907 - 1994

Mark D. Holcomb, MD, a native of Centerview, Mo., died June 1, 1994, in Enid. Dr. Holcomb was raised and educated in Missouri and earned his medical degree from the University of Louisville (Ky.) School of Medicine in 1936. Following his graduation, he completed an internship in Butte, Montana, and a residency at the Montana State Tuberculosis sanitarium. Dr. Holcomb served with the U.S. Army Medical Corps in the South Pacific from 1940 to 1946, rising from the rank of Lieutenant to Lieutenant Colonel and earning a number of decorations and citations, including the Bronze Star. He began his general medical and surgery practice in Enid in 1946. Prior to his retirement in 1983, he held numerous professional positions, including service on the Board of Directors of the Garfield-Kingfisher County Medical Society and as president of the Oklahoma Association of Family Practice. Dr. Holcomb was a Life Member of the OSMA.

(continued)

Richard Warren Loy, MD
1920 - 1994

Richard W. Loy, MD, co-founder of Pawhuska's Loy Clinic (now the Pawhuska Medical Clinic, Inc.), died July 7, 1994. A diagnostic radiologist who earned his medical degree at the University of Oklahoma School of Medicine in 1949, Dr. Loy was born in Stillwater and raised in Guthrie. He joined his brother, Dr. Bill Loy, in Pawhuska in 1950, where he practiced until 1972, when he left to complete a residency in radiology. From 1975 to 1977 he practiced in Oklahoma City and at the Clinton Indian Hospital, later returning to Pawhuska.

Lawrence Sevier McAlister, MD
1905 - 1994

OSMA Life Member Lawrence S. McAlister, MD, died July 19, 1994, in Norman. Prior to his retirement in 1984, he practiced medicine in Muskogee for almost 55 years. Dr. McAlister was born in Webbers Falls, Indian Territory and first moved to Muskogee in 1911. He earned his medical degree from the University of Nebraska, Omaha, in 1929, and after completing his internship and postgraduate work in radiology, returned to Muskogee to practice. During World War II, Dr. McAlister, the only physician doing x-ray diagnosis and therapeutic treatment in an area of approximately 100,000 population, was deemed essential in his position and asked to remain in Muskogee.

John Hobson Veazey, MD
1901 - 1994

Dr. J. Hobson Veazey, a 1926 graduate of the University of Texas Medical Branch at Galveston, died July 11, 1994. A native of Van Alstyne, Tex., Dr. Veazey practiced internal medicine in Madill, Okla., from 1929 to 1935. The following year he moved to Ardmore, where he co-founded the Medical Arts Clinic of Ardmore in 1952. He was an OSMA Life Member, a past president of the Carter, Love, Marshall County Medical Association, and an active member of his church and community.

In Memoriam

1993

Mark Royal Johnson, MD	July 12
Edward David Greenberger, MD	August 6
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

1994

Fannie Lou Leney Hayward, MD	January 2
Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13
Earl Mathiews Woodson, MD	February 20
Tom Lamar Johnson, MD	March 5
Orville Main Rippy, MD	March 11
Minor Elliott Gordon, MD	March 14
George Loren Norris, MD	March 27
Max A. Glaze, MD	April 29
Winfred Aaron Showman, MD	May 14
Mark Daniel Holcomb, MD	June 1
Carter William Mathews, MD	June 3
Harold Ray Sanders, MD	June 15
Robert Bruce Howard, MD	June 16
Richard Warren Loy, MD	July 7
John Hobson Veazey, MD	July 11
Wesley A. Whittlesey, MD	July 12
Laurence Sevier McAlister, MD	July 19
Jess Duval Herrmann, MD	August 16

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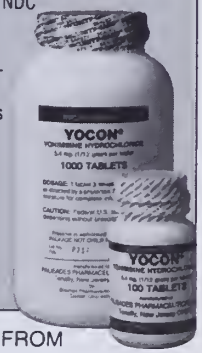

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2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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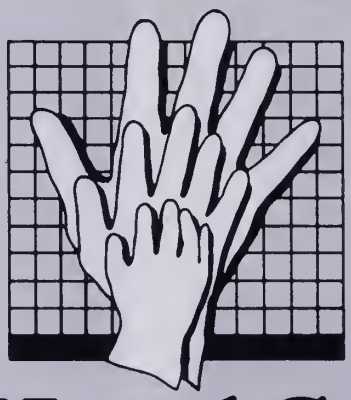

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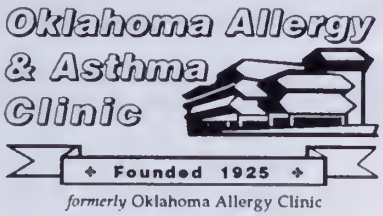

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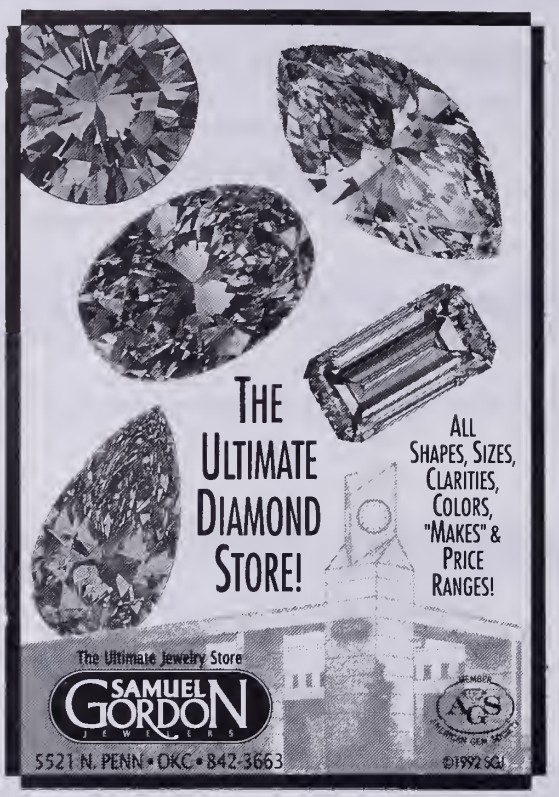

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One Choice/One Voice

"The Power of One," the OSMAA theme for this year, ties in with a recent message from our national president, Barbara Tippins. She is asking AMA Alliance leaders and members to focus on four simple words: One Choice/One Voice. American medicine is gradually being caught in a pincer movement between industry and government. We need to promote unity, solidarity, and shared vision among physicians and physicians' spouses. This we can do by reminding ourselves that when we choose to join, we unite with a stronger force to speak on the issues that will shape our future. It is our window of opportunity, a chance for us to speak with one voice on behalf of all patients and physicians in our communities. If we are to promote the One Choice/One Voice philosophy, we must reach out to all physicians' spouses to say, "Please join us. We need you and you need us." We cannot depend solely on our membership committees to recruit our new members, retain our current members, and renew our former members. Every member must be willing to be involved in making the medical alliance the first organization physicians' spouses join and the last one they leave. When physicians' spouses realize that we care enough to personally seek them out to ask them to join us, they will be more apt to respond in a positive way. I hope that all physicians across the state of Oklahoma will encourage their spouses to join the Oklahoma State Medical Association Alliance.

We need to promote unity, solidarity, and shared vision among physicians and physicians' spouses.

Five alliance members, Cheryl Baker, Karen Mask, Barbara Jett, "K" Caldwell, and I were delegates to the AMAA national convention in Chicago, June 11-15. Also attending the convention from Oklahoma was Susan Paddock, who served last year as national health promotions chair. She has recently been elected to a field representative position. Sherry Strebel, a past national AMAA president and currently serving on the AMA AMPAC board, was a presenter.

This summer the "Pony Express," our leadership seminar, was presented to county officers in Oklahoma City, Enid, and Tulsa. Leaders from surrounding counties were urged to attend. Membership, health promotions, AMA-ERF, and legislative issues were discussed. Our state board members in charge of these areas were the presenters.

—Maggie Hubner
OSMAA President

■ **The Second Annual Oklahoma Regional Medical Conference** will be held at Tulsa's Adam's Mark Hotel on Thursday and Friday, October 27 and 28. This year's theme is "Running the Race: Treatment Perspectives in a Managed Care Environment." The conference will provide both clinical and operational updates to physicians. The Thursday lectures on managed care are for healthcare administrative staff, too. Cosponsors of the event are Oklahoma Community Healthcare Alliance and Preferred Physicians, Inc. For more information, contact Jeff Clark, Oklahoma Community Healthcare Alliance, (918) 488-6057, or Connie Carson-Roark, Preferred Physicians, Inc., (918) 481-7777.

■ **Two years to the day before the O.J. Simpson case focused** national attention on family violence, the AMA's Council on Ethical and Judicial Affairs issued physician guidelines for diagnosing, treating, and reporting domestic violence. Those guidelines included: (1) Routinely ask about abuse as part of a medical history; (2) Become familiar with family violence protocols; (3) Work with community resources; (4) Be Aware of misconceptions about abuse. AMA's National Coalition of Physician Against Family Violence has more than 600 members—double the 1993 number—and serves as a resource for developing community violence prevention initiatives. To join the coalition, call (800) 262-3211, ext. 5066.

■ **Dr. Dennis A. Weigand, professor and vice chair of the** University of Oklahoma College of Medicine's Department of Dermatology, has been named state chairman for the Dermatology Foundation's 1994 Leaders Society Campaign. As part of a nationwide campaign, Dr. Weigand will head Oklahoma's effort to raise funds for dermatologic research.

■ **The Oklahoma Conference on Fetal and Infant Mor**tality: A Community Approach to Mortality Prevention will be held November 3 and 4, 1994 at the Metro Tech Conference Center in Oklahoma City. The conference will bring together all persons concerned with fetal and infant death and will present a community-based process and systems approach to determine how health services can be improved to help prevent fetal and infant death. The two-day program will include address key elements in the process of developing a community-based Fetal and Infant Mortality Analysis Program; the impact of unintended pregnancy and family planning services; community-based interagency, intra-agency, and interdisciplinary collaboration; national and local technical/funding resources; complete birth and death certificate information; and gamily interviewing and grief counseling.

Sara Reed DePersio, MD, deputy commissioner of the state health department's Public Health Services, will introduce the plenary speaker, Henry Theid, MD, of the American College of Obstetrics and Gynecology, for his address "History and Purpose of Fetal and Infant Mortality Review." Dr. Roger E. Sheldon, professor of pediatrics at the University of Oklahoma Health Sciences Center (OUHSC), will deliver the luncheon address on Friday. His topic will be "Where Do We Go from Here?"

Sponsors of the program are the Oklahoma Fetal and Infant Mortality Analysis Program, OUHSC Department of Pediatrics; Oklahoma State Department of Health/Maternal and Infant Health and the Planning and Evaluation Division; and the OUHSC college of Public Health. /for registration information, contact the OU College of Public Health, (405) 271-2342.

■ **Dates for the AMA's 1995 National Leadership Confer**ence are April 23-25. The conference will be at the Washington, D.C., Hilton and Towers. The preliminary meeting schedule includes optional sessions on Sunday morning, April 23, followed by the opening session and a reception. Plenary and breakout sessions are scheduled for April 24 and 25, to be followed by congressional receptions on Capitol Hill the afternoon of the 25th. Medical societies are urged to plan visits with their elected representatives on Wednesday, April 26. For conference information, call (800) 262-3211, ext. 5473.

■ **Despite a 1994 association budget deficit, the AMA will** not increase membership dues for 1995. The shortfall for 1994 will be approximately $23.8 million, according to the AMA board, which attributes the deficit to accounting changes, taxes, and nonoperating expenses—including $4.7 million spent on health system reform activities in addition to the largest single item, a $14.7 million one-time charge for retirement benefits. Nancy W. Dickey, MD, AMA board secretary-treasurer, told the House of Delegates in June that the deficit would "not curtail association reform activities." AMA reserves, which fund AMA health system reform activities, now total more than $125 million—$3 million over budget. In 1994, AMA dues increased $20 to $420—the first increase in five years. Regular members now pay $420 annually; physicians in their first year of practice, $210; in their second year of practice, $315; in military service, $280; residents, $45; and medical students, $20. Members of unified societies (including OSMA) in the first four categories will receive a 10% dues rebate for 1995.

■ **Dr. Steven A. Crawford, Yukon, is the newly elected pres**ident of the Oklahoma Academy of Family Physicians (OAFP) Other officers on the slate are Dr. Pat Bell, Tishomingo, president-elect; Dr. Brent Laughlin, Tulsa, vice-president; Dr. Mike Woods, Ramona, secretary-treasurer; and Dr. Richard Reinking, Tulsa, immediate past president. J

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POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscriptions to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single copies are $3 per copy, prepaid, subject to availability.

Reprints of articles are available either from the authors or from University Microfilms International, 300 North Zeeb Road, Dept. P.R., Ann Arbor, MI 48106, 1-800-521-3044.

National advertising representative: State Medical Publishers Network, 11437 West 106th Street, Overland Park, KS 66214, (913) 888-8781, 1-800-788-2360.

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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The Forty-Three Commandments

Oklahoma again achieved a national ranking in the top 10% of states in the rate of MD physicians sanctioned by their state licensing board in 1994. For many years the Oklahoma Board of Medical Licensure and Supervision has consistently ranked nationally in the upper ten percentile in the rate of disciplinary actions. We may infer from this trend that Oklahoma either has the worst corps of physicians in the nation or the most stringent licensing board in the land. Whichever the case may be, such a statistic calls for thoughtful consideration by Oklahoma's medical profession. Either Oklahoma's medical education standards or the Board's procedures must be re-evaluated.

There are indications that the board plans new actions as, at the board's request, recently enacted legislation (HB 2123) considerably enlarged their legal powers. The board now can make every annual license renewal contingent on "continuing medical education." The nature and definition of "continuing medical education" is not made clear, and any difference in specialty needs for CME is ignored. The law, as written, requires the same continuing medical education of the neurosurgeon and the pediatrician.

Also, the new law empowers the board to... "impose"... "a period of free public or charity service..." on the physician as a disciplinary action. As yet there is no experience with the operation of this section of the new law, but the vague wording raises the specter of the accused physician mandated to treat patients selected by the board. There is grave doubt that a valid patient/physician contract can be established by board order. What is more certain is that no sane patient would knowingly consent to be treated by a physician assigned to the task as a disciplinary action. The intent of the board in seeking these additional powers presently remains shrouded in the cloud of the Olympian mountain top.

The board has recently finalized a contract with the OSMA Medical Services Council for consultative opinions on pending complaints, and this is an initiative that deserves approval from Oklahoma physicians. The complaints to the board quite often involve medical intricacies that are beyond the grasp of the lawyers and staff of the board. Yet *any* action taken against a licensee makes an indelible blotch that haunts the physician forever. ANY licensing board action that becomes part of the public record damages the career of the licensee. Thus it is an important positive step for the board to solicit opinions from the Medical Services Council on medical matters before acting.

Not subject to such approval are the new board amendments to the definition of unprofessional conduct that can lead to license revocation. The new regulations are so full of vague definitions and concepts requiring subjective interpretation that no physician could practice without being in violation. If ever a complaint focuses board attention on any physician, one or many of the Forty-Three Commandments could be interpreted as violated as they are now written. The subjective caprice of the board is left essentially unbridled in the new medical practice act and the new unprofessional conduct rules.

Moses came down from the mountain with Ten Commandments that have guided mankind for several thousand years. The Forty-Three Commandments of the board appear to lack comparable inspiration and insight, and should be reworked for the benefit of the citizens of Oklahoma.

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Our Own Worst Enemy

"A view from the top," the ad reads in describing a lush penthouse for sale. But what relevance do those words have for this President's Page? Let me start by saying that you and I (excluding all past presidents) have significant differences in our perspectives. I'm not making any judgments as to whose perspective is correct, but merely stating that they are quite different.

Working all those years at the local and county levels, and then in the House of Delegates, gave me the same perspective that many of you possess. The critical and sometimes pessimistic view that "the leadership really doesn't know what it is doing," or the perception that "I know I could do a better job" dominates the most common train of thought. But now that I am in this position because of your trust and support, I can tell you that what I see every day is not pretty. For now I am the brunt of all the criticism and false impressions so widely held by many members of our association, including myself at one point in time.

I once thought that working on your behalf would be an honor, but trust me, it is a lodestone around my neck. My fax machine hums with all the correspondence many of you send to me or to other members of our society. Often those communications are full of untruths, innuendoes, and blatant misstatements of the fact. For what purpose? Are some of you so threatened by current events that you feel you must lash out irresponsibly against your own colleagues? Or do you feel the need to create a crisis to divert attention away from the skeletons in your own little closets? What effect do you wish your demeaning and often degrading remarks to have upon your colleagues? These are the same tactics used so successfully by the Clinton administration, and when I see such tactics at use in our profession it causes me much pain. You've heard it before, but it is worth repeating: "We are our own worst enemy."

When I read or hear of attacks upon our own OSMA or the AMA, I must remind physicians that if you don't like the system, introduce resolutions to change it. OSMA members must remember that ours is a democratic process, and even though certain physicians may feel strongly about a particular issue, it is incumbent upon them to convince the rest of us that their position is in the best interests of all our members.

Another responsibility of this office is a seat on the Board of Directors of PLICO. I also chose to sit on PLICO's Claims Committee. Again, trust me, what I see is often not easy to accept. If you have ever served time on your hospital QA/UR committee, you know of what I speak. At the PLICO level it comes down to how much those events actually cost each of us in dollars and cents.

Dr. Robert McAfee, president of the AMA, recently told the Tulsa County Medical Society, "If we lose the way in which we treat each other, then we've lost it all." We have been so busy trying to protect the patient-physician relationship that we have completely overlooked the even more sacred physician-physician relationship. Manipulators from within and without have very effectively driven large wedges into the relationship. And I agree with Dr. McAfee that if we don't take immediate steps to correct these wrongs, then we will have lost it all.

One of the greatest disappointments I must witness each day is the unwillingness of physicians to participate in the political process. A close colleague was quoted as saying, "There are two things I never discuss with patients—religion and politics." This philosophy makes no sense. I'm not suggesting that we sermonize from the examining room or ask for campaign contributions at the window. However, I propose to you that discussing such issues with your patients will only strengthen the patient-physician relationship. I would ask all OSMA members to get involved immediately in those political races of interest to you in your own localities. A few hours of a physician's time and a few dollars to a politician's war chest will reap great benefits for medicine in the months ahead. By the time you read these words I'm sure there will have been much bloodletting on the campaign trail, and your presence may very well be needed—as well as appreciated—come late October.

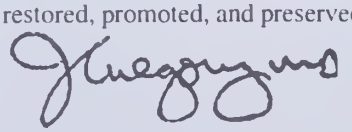
Lastly, the continued takeover of the practice of medicine by corporate America is very alarming. Many of you are becoming increasingly associated with behaviors that we detest in society, those commercial and self-protective behaviors. As Dr. James Todd said, "I just hope that physicians realize what they are doing. They could be ruining their careers for the next 30 or 40 years."

As the battle over the future of the health care delivery system continues, the decisions you and I make today will truly affect the rest of our lives. The central issue is: Who will control the health care system? Do *you* want to control the system, or do you wish to give the control to bureaucrats and administrators?

In response to our challenge of a recent request for proposal (RFP) let in Oklahoma and Tulsa counties, physicians were told that "many well respected and eminently qualified physicians have contacted us in an attempt to cut a deal for themselves to become sole providers in our plans." Why are so many of our "well respected and eminently qualified" colleagues so eager to stab the rest of us in the back? Dr. McAfee is correct.

I guess I am too idealistic about what it means to be a physician. I feel that it is truly an honor and a privilege to serve my fellow human beings. It is such a pity that not all of us share or exhibit these same ideals. It is time for the silent majority to rise up and take control of the situation. Physicians must break out of that shell of pseudo-isolationism that they have built around themselves in their day-to-day practice of medicine and do what is right for themselves and their profession. We must not allow individuals to cut deals for themselves at our expense. The whole is greater than its parts, and the Art and Science of Medicine must be restored, promoted, and preserved.

Good Day.



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Type 2 Cryoglobulinemia and Hepatitis C Virus: Its Recognition and Treatment

Abdul Nadir, MD; James W. Smith, PhD; Billy Matter, MD; Dennis Mask, MD; Ronald Gilcher, MD; David H. Van Thiel, MD

Type 2 mixed cryoglobulinemia is a relatively common although rarely recognized consequence of chronic hepatitis C virus infection. Its detection should be pursued in individuals with lower extremity vasculitis which occurs in association with other signs of systemic disease such as proteinuria or a peripheral neuropathy.

Importantly, HCV-associated cryoglobulinemia can occur in individuals with clinical evidence for cryoglobulinemia but without any evidence of detectable liver injury.

Two cases recently seen in Oklahoma demonstrating these points are reported.

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, and the Oklahoma Blood Institute, Oklahoma City, Oklahoma.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

MIXED CRYOBULINEMIA (CG) is an immunoproliferative disorder characterized by the presence of cryoglobulins which are cold-precipitable immune complexes having rheumatoid factor-like activity. A typical triad of clinical manifestations consisting of purpura, weakness, and arthralgia is present in most cases.[1-3] Cyoglobulinemia can be classified into three distinct subtypes. Type 1 cryoglobulinemia consists of monoclonal immunoglobulin complexes without rheumatoid factor-like activity. Type 1 cryoglobulinemia is usually associated with a malignant condition of the immune system. Type 2 cryoglobulinemia is characterized by immune complexes consisting of polyclonal immunoglobulin G and monoclonal immunoglobulin M rheumatoid factor. Type 3 cryoglobulinemia is characterized by the presence of both polyclonal immunoglobulin G. and polyclonal IgM and/or IgA rheumatoid factor.[1]

Recently, a possible etiopathogenic role for hepatitis C virus (HCV) in mixed cryoglobulinemia type 2 and type 3 has been suggested as a consequence of their coincidence in several different disease populations[4-16] In a case control study, Agnello et al. reported that 16 (84%) of 19 patients with type 2 cryoglobulinemia were positive for HCV-RNA by polymerase chain reactions (PCR).[7] Both HCV antibody and HCV-RNA were noted to be concentrated 10- and 1000-fold, respectively, in the cryoglobulins examined.[7]

The vasculitis that occurs with cryoglobulinemia usually presents as a painful purpuric skin lesion usually on the distal lower extremities. It can involve other organs such as the kidneys, liv-

er, gastrointestinal tract, peripheral nerves, and heart.[2] In an attempt to halt the progression of the tissue injury associated with type 2 mixed cryoglobulinemia, various therapies including corticosteroids and cytotoxic drugs have been given to patients with little or no overall benefit.[2,17-20] In cryoglobulinemia associated with HCV viremia, immunosuppressive drugs can exacerbate the problem by enhancing the replication of the hepatitis C virus. Interferon α (IFN) by eradicating HCV viremia may be the ideal therapy for cryoglobulinemia associated with HCV.

Herein are reported two cases of vasculitis associated with cryoglobulinemia and hepatitis C viremia. Both plasmapheresis and IFN were tried sequentially to reduce the adverse consequences of cryoglobulinemia in case 1. In case 2, IFN alone was utilized to treat the underlying viral infection infection and eliminate the cryoglobulinemia.

Figure 1. Rash on patient's legs consisting of rust-colored pinpoint lesions and a heavier brown reticular pattern on the feet.

Figure 2. Bilateral diffuse lower extremity petechial rash proximally merging into a confluent erythema distally. Note swelling of legs as compared to the fingings in Figure 1.

Case Reports

Case 1.—A 52-year-old women was referred to the investigators for evaluation and management of her end-stage liver disease. She was found to be HCV-RNA positive by PCR. Presumably, she acquired HCV as a consequence of blood transfusions received 14 years earlier. Two years prior to her referral, she had an upper gastrointestinal bleed which was documented endoscopically to be from gastric varices. She was treated with the surgical creation of a splenorenal shunt (SRS). Since her SRS, she has experienced severe fatigue, continued swelling of her legs, and recurrent episodes of poorly localized abdominal pain and arthralgias. For these various complaints, she had been treated with a variety of diuretics and analgesics without success.

Her past medical history was positive for the presence of diabetes mellitus requiring insulin, mild renal insufficiency, a symmetrical distal peripheral neuropathy, small vessel occlusive disease of the brain, and hypertension. Her physical examination was significant for the presence of icterus, ascites, peripheral edema, asterixis, and a bilateral lower erythematous extremity rash as shown in Figure 1.

The various laboratory investigations she was subjected to demonstrated a macrocytic anemia, thrombocytopenia, hyperbilirubinemia, a mild pan-derangement of her liver injury tests, and renal insufficiency. The results of these investigations are summarized in Table 1. When tested for the presence of cryoglobulins, her cryocrit was reported as 20%. Subsequently, she was found to have an IgM monoclonal spike in her protein electrophoresis, a positive serum rheumatoid factor at a titer of 1:5120, a C3 level of 28 mg/dl, a C4 level of less than 8 mg/dl and a CH50 level of 3 (the latter 3 document complement consumption via both the intrinsic and extrinsic systems).

An immunoelectrophoresis of her cold saline washed cryoprecipitate revealed the presence of 2 IgM (K) cryoglobulins, HCV-RNA and HCV-Ab. A diagnosis of type 2 cryoglobulinemia with cutaneous vasculitis, peripheral neuropathy, cerebral vasculitis, glomerulonephritis, hypertension, and visceral abdominal pain associated with HCV viremia was made.

During her hospitalization, spontaneous exacerbations of her lower extremity cutaneous vasculitis and purpura were noted. In an attempt

to reduce the levels of circulating cryoglobulins in her blood to control her symptoms, plasmapheresis was initiated. Pre- and post-plasma exchange cryoglobulin levels were determined and documented disappearance of all identifiable cryoglobulins immediately after the plasmapheresis. Unfortunately, within 48 hours her cutaneous purpura recurred and an easily measurable cryocrit was detectable. The frequency of her plasmapheresis treatments was increased to three times a week and was followed by a continuous exacerbation of her purpura. At this point interferon α2b (IFN) at a dose of 3 million units per day was added to her schedule of 3 plasmapheresis sessions per week. A slight improvement in the purpura and tenderness over her legs and knee joints was noted. During a five-day period when the IFN was discontinued due to severe thrombocytopenia, her plasmapheresis schedule was increased to occur daily. A severe exacerbation of her purpura associated with overt leg tenderness and bilateral ankle and knee arthralgias developed (Fig. 2). As a result, the plasmapheresis sessions were discontinued and she was restarted on IFN 3 million units daily. The purpura disappeared on IFN treatment and recurred whenever the IFN was discontinued for more than 2 to 3 days.

Case 2.— This 35-year-old white male was in excellent health until three months prior to admission when he developed a viral syndrome. This was followed by the recognition of hypertension (230/180) and proteinuria. Biochemical studies demonstrated a serum creatinine of 1.4 mg/dl, an albumin level of 2.1 g/dl and 4+ proteinuria.

On a review of his past medical history, he admitted to several years of drug abuse and upon questioning, the development of episodic petechial hemorrhages involving his lower legs.

A renal biopsy was obtained and was interpreted as being consistent with either Wegener's granulomatosis or cryoglobulinemia. He was subsequently demonstrated to have high grade cryoglobulinemia with a cryocrit of 30% and to be anti-HCV positive.

His physical examination was entirely unremarkable except for hypertension. His creatinine clearance was calculated to be 146 ml/min and his 24-hour urinary protein loss was determined to be 5.4 g/24 hr.

A liver biopsy was obtained, despite normal liver enzymes, and revealed a mild lobular hepatitis having a Knodell score of 3 (range of 0-22).

The patient was started on IFN therapy at a dose of 5 MU daily and has experienced a marked reduction in his proteinuria and cryocrit determinations, as well as episodes of petechial bleeding into the skin of his lower legs.

Table 1. Laboratory Investigations Obtained in Case 1

Parameter	Patient Result	Normal Values	Units
WBC	2.8 L	4.0-10.4	1000/MM³
RBC	3.19 L	3.90-5.10	10⁶/MM³
Hemoglobin	11.9	11.0-15.0	G/DL
Hematocrit	5.0	34.0-45.0	%
MCV	109.7 H	82.0-99.0	H
MCH	37.3 H	27.0-34.0	rs
MCHC	34.0	33.0-35.0	g/dl
Platelets	45 L	150-433	1000/MM³
BUN	29	5-25	MG/DL
Creatinine	2.0	0.5-1.4	MG/DL
Total Bilirubin	3.0 H	0.4-1.5	MG/DL
Albumin	3.0 L	3.5-5.0	G/DL
Alkaline Phosphatase	94	40-130	IU/1

Discussion

The first case is of interest because it demonstrates three important points about the treatment of cryoglobulinemia associated with HCV viremia. First, plasmapheresis can transiently clear the plasma of circulating immune complexes and cryoglobulins but is followed by a rebound increase in detectable immunoglobulins and symptoms related to the presence of cryoglobulinemia. Second, interferon, by reducing the circulating levels of the inciting antigens and suppressing B cell production of antibodies, lowers the plasma level of cryoglobulins and reduces immune complex formation, ameliorating the clinical consequences of cryoglobulinemia. Third, the reduction in plasma content of cryoglobulins in this case did not directly parallel the objective clinical improvement observed in the patient's purpura. This observation is a consequence of the fact that both plasmapheresis and IFN reduce the level of circulating CG but have no effect on the local inflammatory response to extravasated immune complexes and subsequent cytokine cascade.

Despite the disappointing effect of plasmapheresis in the first case of this report, plasmapheresis may still have a role in the immediate preoperative treatment of patients with HCV-associated cryoglobulinemia who have very high cryocrit. Moreover, the use of continuous intraoperative plasmapheresis may be useful as a means of avoiding the complications of cryoglobulinemia that can occur during surgery as a con-

The use of continuous intraoperative plasmapheresis may be useful as a means of avoiding the complications of cryoglobulinemia that can occur during surgery.

Several case reports and small clinical series have demonstrated the usefulness of IFN in cases of mixed CG associated with HCV viremia.

sequence of operative hypothermia and intravascular immune complex precipitation.

The second case is important as it demonstrates that symptomatic cryoglobulinemia with the nephrotic syndrome can occur in patients with HCV in the absence of any detectable abnormalities of standard liver injury tests. Moreover, it demonstrates that IFN therapy by eliminating the HCV viremia, eliminates the cryoglobulinemia, resulting in a reduction in urinary protein loss and clinical evidence of cryoglobulinemia.

Plasmapheresis is used frequently as a means of eliminating autoreacting antibodies and circulating immune complexes in several different disease processes.[24] Occasionally, the rapid removal of immune complexes and autoantibodies by plasmapheresis can paradoxically enhance autoantibody production. This presumably occurs as a consequence of increased B cell production of such antibodies after plasmapheresis.[25,26] Thus, B-cell inhibiting immunosuppressive agents are often prescribed immediately after plasmapheresis in patients with severe systemic lupus erythematosus,[27] Goodpasture disease,[28] pemphigus vulgaris,[29] and an essential cryoglobulinemia occurring in combination with a circulating neutropenia.[30]

Interferon alpha 2b is a cytokine that has several different mechanisms of action.[31] These include antiproliferative, immunoregulatory, and antiviral effects. These actions of IFN are used clinically to treat chronic viral hepatitis, various neoplastic diseases, and a variety of lymphoproliferative disorders such as hairy cell leukemia and chronic myelogenous leukemia.[32] Because IFN can induce HCV clearance as a result of its antiviral and immune-modulating activities, anecdotal clinical trials of IFN for cryoglobulinemia associated with HCV infection have been reported. Several case reports and small clinical series have demonstrated the usefulness of IFN in cases of mixed CG associated with HCV viremia.[6,11,14,16,21-23,31] Presumably, the beneficial effect of IFN in the treatment of type 2 cryoglobulinemia associated with HCV viremia is the result of its antiviral as well as its antiproliferative actions. The antiviral effect of IFN reduces the stimulus for antibody production and the level of circulating ligand available for immune complex formation. The antiproliferative effects of IFN may suppress the proliferation of CG- producing B cells.[33]

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The Authors

Abdul Nadir, MD, is a fellow in transplant medicine at the Oklahoma Transplantation Institute (OTI), Baptist Medical Center of Oklahoma (BMCO), in Oklahoma City.

James W. Smith, MD, PhD, is associate medical director of the Oklahoma Blood Institute (OBI), Oklahoma City, and oversees plasma exchange therapy performed in Oklahoma City hospitals by OBI.

Billie Matter, MD, is a clinical profesor of medicine at the University of Oklahoma Health Sciences Center and also is in private practice in Oklahoma City.

Dennis Mask, MD, is in private practice in Oklahoma City, specializing in nephrology.

Ronald O. Gilcher, MD, is president and CEO of the Oklahoma Blood Institute. Prior to coming to Oklahoma City, he was medical director of the Central Blood Bank of Pittsburgh, Pa.

David H. Van Thiel, MD, is director of transplant medicine and director of the Hepatology and Digestive Disease Center at BMCO in Oklahoma City.

Introduction to Smart Card Technology and Initial Medical Application

Gary Quick, MD

Smart card technology is the name applied to the use of a plastic card with an embedded computer chip. Recent development of smart card software has allowed storage and retrieval of medical information, affording the opportunity to provide a standardized, portable, accessible medical record for use in prehospital and emergency department patient encounters. We describe the smart card concept and its initial deployment in a section of a large Midwestern urban area.

Direct correspodence to Gary Quick, MD, Section of Emergency Medicine, P.O. Box 26307, Oklahoma City, Oklahoma 73126.

AS CHANGE ACCELERATES in the health care sector, many new techniques and technologies are emerging to assist practitioners and patients alike in dealing effectively with the yet ill-defined system of health care in which we will practice in the future. The purpose of this article is to introduce emergency physicians and other practitioners to a computer technology known as the "smart card," which has recently been applied to personal medical records to facilitate standardization, portability, and accessibility while maintaining security. Oklahoma City has become the first U.S. metropolitan area to apply smart card technology to citywide emergency health care services and record-keeping. The "+MediCard"™ system developed by Advantage Data Systems (ADS), a smart card vendor in the Oklahoma City area, provides local emergency medical personnel immediate access to vital patient information at the site of a medical emergency.

"Smart card" technology integrates a plastic card the size of a credit card with an embedded computer chip containing the patient's pertinent medical information. Card readers located in emergency response vehicles and in hospital emergency departments (ED) allow instantaneous data retrieval of all medical information contained in the card memory. ADS developers anticipate that access to an immediately available personalized medical database should permit emergency medical personnel to respond to the patient's condition in a more informed and, therefore, more effective fashion.

Smart card technology is actually a mature

technology, having been developed by Gemplus of Gemenos, France in the 1970s . The cards and readers have proven to be reliable, relatively standardized, and economical in their utilization in telecommunications, banking, defense, and other industries worldwide. The Gemplus organization has U.S. offices in Gaithersburg, Md., and presently manufactures approximately 14 million cards per month for these industries. Card memory capacity has been increasing rapidly, from 3 kilobytes (K) in the summer of 1993 to a current 16K card. A 32K card may be available as early as mid-1994. Currently patient confidentiality is secured by an encryption process as well as by having the patient alone determine the information to be placed in the card database.

ADS has built upon smart card technology to develop four computer software programs for the +MediCard, among which are a mobile interface and interfaces for the ED, data entry, and pharmacy functions.

The system consists of three major components: the smart card, a palmtop computer, and a personal computer (PC). The +MediCard, a Gemplus smart card with updatable memory (Gemplus GFM4K Serial 1C Memory Card with 4K of EEPROM memory, Gemplus Card International Corp., Gaithersburg, Md.) contains the individual patient medical information. The palmtop reader for use in the ambulance is a Hewlett-Packard 95LX palmtop PC equipped with a Gemplus GPR adaptor to set into the PCMIA slot. Each hospital has a Gemplus GCR200 Smart Card Reader attached to the serial port of an IBM-AT-compatible computer in the ED.

CURRENTLY THE ADS database includes the data categories as depicted in the sections of Figure 1, a portrayal of the 3K smart card initially produced. As in other computerized data systems, memory consumption is based upon predictability of data. Hence, demographic information such as name and address consume much more memory than predictable items such as medications and diagnoses. With the recent expansion of card memory, the database may soon expand to include electrocardiogram and other data which previously have required more memory than the 3K smart card allowed.

Benefits of the up-to-date, accurate patient history available through smart card technology at the time of a medical encounter may be expected to achieve cost savings in many ways:

1. Healthcare practitioners in various locations and practice environments will be linked to critical medical information needed to implement appropriate emergency treatment.

2. Availability of a standardized database at every medical care encounter can bring efficiency to many repetitious processes and prevent inappropriate or redundant testing or treatment.

3. The recognition and prescription of medications can be accomplished more accurately.

4. Pharmacists may recognize and intervene if medication conflicts exist or are contraindicated in such conditions as pregnancy.

5. A new sense of partnership among patients, physicians and other members of the health care team may be promoted.

6. Physicians and other providers may be prompted to carry out prophylactic and preventive measures when accurate knowledge of the patient's condition is available at the encounter. Reminders can be generated in a timely manner for each practitioner participating in the patient's care.

> The smart card clearly offers accessibility and portability of data combined with a secure mechanism to preserve individual privacy and confidentiality.

Applications of smart card technolo-gy potentially include many attractive advantages in the present climate of cost concern and health care reform. The smart card clearly offers accessibility and portability of data combined with a secure mechanism to preserve individual privacy and confidentiality. At the present time, the patient determines what data is entered on his personal card. The convenience of replacing the incessant requirement to fill out forms by an electronic process that is both rapid and accurate is attractive to all parties involved in the care transaction. With one pass of the +MediCard through the card reader, demographic information may be downloaded into a registration computer at the point of care. Data from each sequential encounter can also be added immediately by writing key elements of the encounter to update the card at the point of care. The updated record in its entirety will then be immediately available to the next provider.

On the healthcare reform front, legislation to help automate the healthcare system will likely pass Congress by session's end is the message beginning to be heard from both industry and government sources. Automation legislation may include logging more patient data electronically or

Patient: JOHN O. PUBLIC +MediCard Update 8/23/94

Hospital Pref: DEACONESS
Date Medically Verified (Y)
Living Will (Y)
Blood Type: O+ Organ Donor (Y)
Hospitalized: SOUTHWEST MED. CTR.
Date: 05/20/91
Note: CONTACT DR. SMITH PG 555-6247
IF SEIZURE OCCURS

----------Allergies----------
1) PENICILLINS
2) BARBITURATES
3) INSECT STINGS
4)
5)
6)
7)
8)
9)
10)
11)
12)
13)
ALLERGIC RESPONSE TO AMOXICILLIN

----------Diagnoses----------
1) EPILEPSY
2) ASTHMA
3) BRONCHITIS
4) UNSPECIFIED GI DISORDER
5)
6)
7)
8)
9)
10)
Others: Please ask patient
ESOPHAGEAL REFLUX

----------Emergency Contact----------
JANE A. PUBLIC
(405) 555-5550 Work: (405) 555-5605

----------Physicians----------
1) General Practice
JOHN SMITH, M.D. (405) 555-7676
2) Neurology
SUSIE JONES, M.D. (405) 555-6247
3) Gastroenterology
BOB JEFFRIES, M.D. (405) 555-7676

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Expires: 08/23/97
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Date of Birth: 01/10/1961 Sex (M)
11032 QUAIL CREEK RD
OKLAHOMA CITY, OK 73120

Home Phone: (405) 555-5550
Work Phone : (405) 555-5605

----------Procedures----------
1) NEPHROLITHOTOMY (KIDNEY STONE) 03/83
2) INGUINAL HERNIA REPAIR 08/76
3) UNSPECIFIED ESOPHAGUS PROCEDURE 09/93
4)
5)
6)
7)
8)
9)
10)

EXPER. SOME PROBLEMS W/ ANESTHESIA

----------Medications----------
1) CARAFATE TAB 1GM: ACHS
2) TEGRETOL 200MG: TID
3) BENADRYL 25MG: O4H
4) SLO-BID 300MG: 1BID
5) PROVENTIL AER: 2OID
6) ATROVENT INHALER: 1TID
7) INTAL INHALER: QID
8) ORGANIDIN 30MG: 2OID
9) VANCERIL: 1TID
10)

CHECK THEOPHYLLINE BLOOD LEVEL MONTHLY

----------Insurance----------
Guarantor: JOHN O. PUBLIC
Soc Sec: 441-68-3308 ( )

Policy: Y441-68-3308 Grp: 700000
AMER. PHY. INS.
120 CHERRY ORCHARD
OKLAHOMA CITY, OK 73120
(405) 555-1244 Cert: (800) 555-5656

----------Secondary----------
( )
Policy: Grp:

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Figure 1. Portrayal of a smart card database.

plugging Americans into insurers and hospitals by means of plastic cards. Yet to be resolved is the issue of whether the automation should broadly embrace a centralized database employing mainframe computers or decentralized databases at the personal or hospital level .[1]

Other countries may be somewhat ahead of the United States in the deployment of smart card technology for healthcare applications. Trials with card systems have been initiated in France and the United Kingdom.[2] Health cards are being introduced for the populations of Portugal, Spain, West Germany, and the Canadian provinces of Saskatchewan and British Columbia for the purpose of patient identification; currently most systems do not include medical information. Independent analysts have stated that patient-held computer-readable medical cards will be one of the major growth areas of the 1990s.[3]

The initial deployment of the +MediCard began in late 1993 when ADS offered the system to an area of Oklahoma City with approximately 100,000 residents. The population is served by the Midwest City Regional Hospital ambulance service and includes six area health care centers: Midwest City Regional Hospital, Southwest Medical Center, Presbyterian, Hillcrest, University, and Children's hospitals.

This particular startup plan includes card-reading computers in emergency vehicles and in each of the six hospital emergency departments. When a patient summons an ambulance or arrives at a

participating ED, the card is inserted into a computer either in the emergency vehicle or in the ED proper. Emergency personnel gain immediate access to the patient's medical history, even if the patient is incapacitated by illness or injury.

Eight area pharmacies serve as smart card service centers at which information on the card may be updated at the patient's request at no charge using hardware which stores data on the +MediCard. During the initial trial no patient data is entered at the participating EDs, although future studies will include ED data entry to the card. The plan includes no provision to "telecommunicate" data from emergency vehicles to hospitals, although ambulance personnel may often relay such information by radio after reading data from the patient's +MediCard.

FUNDING HAS BEEN requested for a multicenter trial of +MediCard involving the prenatal obstetrical services of Southwest Medical Center and University Hospital in Oklahoma City. The objectives of the +MediCard prenatal study are to provide an improved management system for obstetrical patients, to demonstrate the economic impact of smart card technology upon obstetrical practice, and to provide a platform for future development of a smart card system that may meet the needs of a wide segment of health care practitioners.[4] This project will rigorously evaluate outcomes such as economic savings and improved quality of prenatal and obstetrical care to women whose prenatal database will be kept current, accurate, and easily accessible to all providers within the limited healthcare study model by means of smart card technology. Currently, prenatal data is gathered at several locations such as physician's office, laboratory, radiology department, ED, and labor and delivery suite. Data acquired at each location is often linked slowly, if at all, to be merged into the patient's record. Consequently, relevant medical history and physical examination data may be absent from the record at a given encounter. Laboratory and ultrasound examinations must sometimes be repeated since the results may not be contained in the portion of the record available to the provider at the time of encounter.

The applications of smart card technology to medicine may have an imminent and far-ranging impact upon the future practice of medicine, especially with respect to the immediate and efficient availability of accurate patient information. Such innovation may rapidly transform the ED environment in which we currently practice into an environment in which a higher percentage of diagnostic and treatment decisions are based upon valid information provided by the smart card at the time of patient encounter. Whether national healthcare reform leads to the wholesale deployment of smart card technology in the form of a national database, or smart card technology finds its niche as a more narrowly employed tool for improving both the quantity and quality of information available at the time of encounter, it appears that smart card technology may play a significant role in our practice in the near future.

Conclusion

Smart card technology is a mature technology which has recently been applied to development of a standardized, portable, accessible medical record. The smart card has potential application as a component in the development of an electronic record. Feasibility and outcome studies are being planned to evaluate limited deployment of the smart card.

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The Author

Gary Quick, MD, is associate professor of surgery and chief, Section of Emergency Medicine, in the Department of Surgery at the University of Oklahoma Health Sciences Center.

Wiley McCollum, Oklahoma's first board certified cardiologist, used to tell medical students that the most valuable part of his training was the time he spent working in a Waynoka grocery store...

LEADERS IN MEDICINE

Waynoka Wisdom

BY RICHARD GREEN
PHOTOGRAPHY BY VICTOR R. RIVAS

IN HIS GRAVELLY VOICE, he is holding forth from his Laz-y-Boy, behind the TV tray cluttered with papers, a glass of water, a telephone, and objects that if spread out woul d almost cover a regular sized desktop. But like a lot of retired elderly gentlemen, Dr. Wiley T. McCollum has consolidated and reduced the size of life's day-to-day essential ingredients.

His old stethoscope is in the room, but not on the tray; the instrument that he pressed to the chests of thousands of patients over 40 years has been a relic since he retired in 1984.

He and Vivian, his third wife, live in a large house in the Oklahoma City suburb of Nichols Hills, but his easy chair and TV tray are command central. They are located at one end of his den in a spot that he and Vivian facetiously call his office. Down the hall, Vivian has a real office with desk, filing cabinets, computer and printer, a copier, and so on.

McCollum in 1942, when he was serving in the Pacific.

McCollum is 78 and looks it. With sun-damaged skin and a voice that can get so hoarse and scratchy that the sound of it suggests grinding glass, or pain. He moves around arthritically, on the cusp of frailty, but still manages to play golf periodically.

Since he started "falling apart" during his fifties, he has endured more than 20 major surgeries including eyes, knees, hips, and various locales in and around the abdominal area. He believes some of his medical problems resulted from exposure to the x-rays he used in a cardiac catheterization lab.

There is no one locally with whom to compare his experience. He was the first and only board certified cardiologist working in the first and only private lab in the state for much of the 1950s. But he says his colleagues

directing similar labs in other states all suffered from cataracts during their fifties and apparently many of them, like him, have had severe degenerative joint problems.

As his physical problems worsened during the last decade of his practice, he suffered a series of personal crises that could have broken his spirit if not his mind. But as his friends and former medical associates attest: Wiley McCollum is incredibly tough and extraordinarily resilient.

He believes these qualities were mainly learned through his experiences while growing up poor in the spartan existence of northwestern Oklahoma—where the elements seemed to be in charge. The land literally blew away during the Dust Bowl years, and his father's cattle suffocated in a canyon during a blizzard.

Down the hall from his den is a room where McCollum stores some of his memorabilia—plaques and framed awards on the wall, gifts from patients, family photos, that sort of thing. In the center of the room is a large octagonal oak dining room table. It means more to McCollum than all the rest of the awards and mementoes put together, for it is the McCollum family table from his childhood in Waynoka during the Twenties.

The table can fuel his memories in powerful ways. He can see the family still, his mother and father, brothers and sisters, sitting around the table at dinner. He can hear the Union Pacific or the Santa Fe Super Chief chugging through town bound for California or Chicago or New York. He can remember the kerosene lantern, just there in the middle of the table, illuminating schoolwork in the evenings...

...The table was the axis around which the McCollum family revolved and reconvened at dinnertime until the older ones moved away. When Wiley was born in 1916, one of his brothers was already dead and two others were in high school. He was the last of eight children born to John and Carrie McCollum.

He was born at home on a farm south of Waynoka. It was a long, difficult birth, a foot presentation, and Carrie bled a lot. The doctor had been called, but he had been detained. So midwife Bessie Dye presided. She allowed gravity to help some by putting Carrie on an ironing board tilted downward. In due course, Wiley McCollum appeared but apparently not without one hell of a fight.

His ancestors on both sides of the family were from Ireland; the McCollums near

McCollum, left, was a member of the Waynoka High School Brass Quartet. Rounding out the group are (l to r) Frances Donnell, Don Thornhill, and Rex Dunn.

Belfast and the Wileys near Dublin. Wiley's parents met in Kansas and were married in 1894. Carrie had graduated from the eighth grade and was teaching school; she also taught her husband how to read and write.

Ten years later John and Carrie left Ft. Scott, Kan., in a covered wagon and settled in a section of the future state of Oklahoma called No Man's Land. They bought land south of the Cimarron River in Major County and John farmed that land with help from his older sons.

Two of them, Walter and Estel, developed typhoid fever. Though both recovered, Estel had been profoundly impressed by the doctor and the power of the pink pills he had administered. He announced he had found his profession: medicine.

NO ONE LAUGHED. Although the odds were against a poor boy becoming a doctor, John and Carrie encouraged Estel and said if he worked hard he would make it. "My father told us we could be anything except railroaders," Wiley says.

After losing his entire herd—80 head—to the blizzard in 1919, John McCollum moved into Waynoka with the rest of the family and, like most of the townsfolk, got a job working in the railroad yard. He retired from the railroad at age 66 in 1940, the same year Wiley graduated from medical school. It was no coincidence that the father kept working until his last child had finished his education. Speaking proudly for both Carrie and himself, John told the president of the University of

As his friends and former medical associates attest: Wiley McCollum is incredibly tough and extraordinarily resilient.

Oklahoma at the commencement ceremony, "We've been to college for 37 years."

* * *

As McCollum matured, he understood that [Dr.] Clapper wasn't a miracle worker but a man of civility, compassion, and intelligence.

AS A CHILD, Wiley did odd jobs for spending money, but he didn't go to work in earnest until he was 12 in 1928. The day after he graduated from the eighth grade, he got a job at the Hutchison Central Market.

Since making deliveries in the Hutchison brothers' 1924 Model T Ford was part of the job, Wiley had to answer, yes, that he knew how to drive, in order to get hired. Then, he somehow learned because he had to... so he could work 83 hours a week for $5.55. That, McCollum says, worked out to 6 $^{2}/_{3}$ cents an hour.

He banked $5 a week, drawing 4% interest, and spent the rest, presumably all on Sunday afternoon, the only time off he had. He didn't know exactly what he was saving for, but the importance of savings was underscored when these Scrooges, the Hutchinson brothers, inexplicably just up and gave Wiley that Model T. However, after a few spins he decided he couldn't afford to pay nine cents a gallon for gas after his summer job ended.

McCollum learned good work habits during those mandatory evenings spent studying at the table, and he made good grades. And there were signs that he had an inquisitive mind. One summer, he wanted to make the kitchen cooler for his mother, so he attached a small perforated lead pipe to a water hose so that the water would drip onto some packing material in a hollowed-out apple basket. He placed a fan so it would blow air through the moist packing material and voila! He had produced a water cooler.

The summer that Wiley was 12, his brother Estel was in medical school at OU. And like Estel, Wiley had recovered from a serious illness, and the family perceived that the doctor played an important role in the recovery. When he was in the third grade, Wiley developed lobar pneumonia and after a time became comatose. Although the family physician, Dr. E.P. Clapper, couldn't do much except give aspirin and castor oil, he visited Wiley every day until his recovery was well underway.

While this experience didn't transform Wiley into an aspiring physician, it did increase his admiration for Dr. Clapper. As

Wiley McCollum served at the 204th General Hospital in the Pacific during World War II and, like veterans everywhere, has stacks of photos, souvenirs, and memories that seem like yesterday.

McCollum matured, he understood that Clapper wasn't a miracle worker but a man of civility, compassion, and intelligence. After Wiley decided to pursue a medical career, his role model was the Waynoka general practitioner.

But this would come later. In high school, he was attracted to music and spent his savings, $85, for a Conn cornet. He found he had some talent and practiced all the harder, especially to impress the band director, Glen Varnam. In 1931, Wiley competed at the Tri-State Music Festival in Enid. One of the judges was John Philip Sousa.

His performance level dipped during his junior year when he fell in love with a contralto. When she and her family moved away, Wiley soothed himself by adding another instrument to his repertoire, the cello. At the age of 15, he played in a dance band and once earned the amazing sum of $3 for three hours played.

As a cornet soloist his senior year, he won the district contest playing "When You and I Were Young, Maggie" and he was named co-director of the junior high band by Varnam, whom he venerated. So strong was Varnam's

influence on Wiley that the young man decided to go to college to become a band director. Furthermore, Wiley, at 16, decided to quit smoking so that he would be a good example, like Varnam, to his music students.

Both Wiley and Varnam wound up in Stillwater in 1933. Varnam had been named the high school band director and Wiley was a freshman music student at Oklahoma A&M. His plans and goals had seemed so logical and desirable, and then rather quickly he realized he was in over his head. First, he wasn't as good as he thought; after tryouts he was sixth chair in the trumpet section. Although his main problem, inadequate sight-reading skills, could be remediated with practice, he didn't have time to practice the prescribed hour a day on cornet and hour daily on piano.

McCollum was enrolled in 18 semester hours and was working four hours every evening just to eke by financially. Though the decision to drop music had been relatively easy, it was still jarring. Fortunately, Wiley was coming under the influence of his chemistry professor, Dr. Otis Dermer. "If you can picture Icabod Crain (of *The Legend of Sleepy Hollow),* that was Prof. Dermer," says McCollum. "But he was a brilliant and inspirational teacher who provided both a good introduction to science and the motivation to use the tools to make little discoveries."

For example, when McCollum read in a textbook that putrescine had never been synthesized, he decided to stake out that territory. It's easier to understand the young man's motivation given the fact, he says, that he didn't even know what putrescine was (the compound responsible for the foul smell of stools). "Dr. Dermer gave me some references to get me started, and within a few months I had succeeded in making putrescine." However dubious the achievement, Wiley T. McCollum had recorded a scientific first before his 20th birthday.

Longtime friend Dr. A.C. Lisle shares some WWII tales with McCollum.

By then, he was taking embryology and had attended a pre-med smoker (where he felt obliged to start smoking again). Soon, he was hooked on the idea of becoming a doctor (and on cigarettes). During his senior year, he was elected president of the pre-med fraternity, Alpha Pi Mu. As always, McCollum was a serious student who did well—with one exception.

During his sophomore year, he again fell in love, this time with the freshman queen, and "my grades went down in proportion to the number of dates we had." McCollum bottomed out with a D in bacteriology and the girl wanted to get married.

Seeing his new plans disintegrating, McCollum took pen in hand during the summer break and explained to his girlfriend that he was simply too poor to get married, adding that he would be in school for another six to eight years. When his mother heard the news, she wept with relief.

He was elected to the student senate and was about to enroll for his senior year when his brother, Estel, a surgeon in Detroit, came to Stillwater to convince him to forego campus politics and enter medical school. Although Wiley was torn—he liked being popular—Estel was very persuasive.

* * *

HAVING REPEATED bacteriology, McCollum entered the University of Oklahoma medical school in 1936. Dr. Clapper was so happy and proud that he gave his former Waynoka patient some of his medical equipment. Like most of his classmates, McCollum wanted to be a general practitioner because "you could begin your practice after only a one-year internship and in some states, like Texas, you didn't even need that."

However, by his junior year, his experiences were leading him more toward a career as a diagnostician, as an internist was then called. During his pediatrics rotation, he was struck by the substantial differences in the symptoms of babies and children suffering from rheumatic heart defects. Some were faint, others sounded like a buzzsaw; the more of those little chests McCollum listened to, the more he realized there was a cacophony of different sounds and even feels.

He felt sure these were not all rheumatic problems and that these differences signified a variety of anatomical and functional defects.

What doctors could identify at that time far outstripped what they could treat effectively.

But in the late 1930s in Oklahoma, only autopsies could provide the definitive evidence.

What doctors could identify at that time far outstripped what they could treat effectively. A perfect example materialized as McCollum's first patient during his first clerkship: Mrs. Smith of Hobart, with previously diagnosed angina pectoris. She was on nitroglycerine, which temporarily controlled the pain, but doctors had nothing more to offer save the prevailing bromides of the day: rest, avoid physical exertion and, if possible while living like an invalid, don't worry, be happy.

Although McCollum never intended to become a surgeon, he enjoyed the surgery rotation because he learned so much from Dr. J. Ross Veal.

Still, it was common knowledge that new medicines, such as the sulfanilamides, were being developed, and that the practice of medicine was about to enter a revolutionary new era. In fact, during McCollum's senior year, 1939-40, several different bacterial infections yielded in stunning fashion to the first sulfa drugs used at University Hospital.

The year 1939 became even more momentous for McCollum when he and Myrldean Scott were married. They had met playing miniature golf in 1933, just before Wiley left for college. Later, she majored in fine arts at Kansas University. They kept up with each other; he visited her in Lawrence and she visited him in Stillwater, and gradually their friendship deepened. They were married before his senior year in medical school and her senior year at KU. Since Myrldean didn't want a career, she never finished her degree.

Wife Vivian joins McCollum during a 1985 visit to their cattle ranch in Chattanooga, Oklahoma.

Their honeymoon was spent looking for the best place for Wiley to intern. Since his father still worked for the railroad, the young couple had a pass, which enabled them to travel free to St. Louis; Detroit; Washington, D.C.; and New York City.

The dean of the OU medical school, a former surgeon general, had given McCollum almost all the endorsement he needed to be accepted at Washington's Gallinger Municipal Hospital. (Gallinger, which had been erected to take care of part of the enormous number of Civil War casualties, is now named D.C. General.) They seemed to want him, and he, understanding that some of the nation's best internists practiced there, surely wanted them.

From day one, McCollum was totally immersed in the work; either he didn't know that France had just fallen to Nazi Germany or he didn't think about it enough to recognize the implications. At Gallinger's, he sopped up new knowledge and experiences and was having the time of his life. Under the direction of Dr. Barney J. Walsh, he x-rayed the heart and coronary arteries with a contrast media, and they had an exhibit of their work at the D.C. Medical Society.

Most often, he didn't even notice how tired he was until he got off. On obstetrics, where the service was shorthanded, McCollum's shifts often consisted of 24 hours on and 12 hours off. "Once, I delivered 12 babies between midnight and daylight," McCollum says. "But I enjoyed it and while I was rotating through OB I thought I might become an obstetrician."

He was exposed to a number of new and exciting drugs, such as quinidine, digitalis, and a variety of sulfa compounds, and was positively thrilled when he used one of them to cure a young girl with measles, encephalitis, and pneumonia. She would have died otherwise. These drugs were extraordinarily powerful and sensitive within the therapeutic range. Once, McCollum mistakenly gave five times the customary dose of one experimental diuretic to a dropsy patient. As a result, the patient spent the evening on a bedpan producing 18 pounds or two and one-half gallons of urine.

Although McCollum never intended to become a surgeon, he enjoyed the surgery rotation because he learned so much from Dr. J. Ross Veal. Among many cogent observations, Veal noted that a large number of surgical patients who were required to have prolonged bedrest developed blood clots. Veal also observed that dehydration seemed to be a factor. "This was all new stuff being taught by the man who was doing the original work," McCollum recalls.

He enjoyed all of his rotations except

orthopedics. As the main charity hospital in Washington, Gallinger's had a very large number of tuberculosis patients and many of them had TB of the bone. "About all we could do for those patients was change the dressing, which was constantly oozing pus, and make them as comfortable as possible until they died."

In July 1941, McCollum was named assistant resident in internal medicine, but by then he knew the United States was about to enter the war. Most of his colleagues had had commissions in medical school, and they had been drafted. Most of their replacements, McCollum says, "were draft dodgers with rich, well-connected parents. I don't know whether they were cowards or not, but I couldn't stand to be around 'em, so on August 15, I went home to Myrldean and said, 'We're joining the Army.'"

* * *

THE FOLLOWING APRIL, Lieutenant McCollum was on one of 50 ships zigzagging through the Pacific bound for the Philippines. He was part of the 204th Reserve General Hospital that would treat casualties from the battles aimed at repelling the invading Japanese. When word was received that the Philippines had fallen, the flotilla zigzagged to Hawaii, where McCollum treated TB patients, ran a rehabilitation hospital and, as the manager, transformed a losing baseball team into champions.

"This bunch had an 18-game losing streak and I told the colonel why, McCollum says. 'They don't even look like a team,' I said. 'They need uniforms and proper equipment.' The colonel, who always carried a swagger stick, waved it and said, 'You're right, McCollum. Go downtown and get whatever you need.'"

To the colonel's chagrin, McCollum spent $2,200 of company funds to get his players looking like a team and then on the strength of three good pitchers and practice, practice, practice, the team started winning until the winning streak matched their previous losing streak. One other factor accounting for the team's success was the fact that they stayed together while their opponents were from units just passing through, on their way to the war.

Finally, the 204th was ordered to Guam, where Capt. McCollum and his baseball team (and a few others) built their hospital by erecting 258 tents in a jungle that had been cleared for that purpose. The tents were 40 by 20 feet with eight to 10 patients per tent. In his ward of five tents, and later in a ward made from plywood by the Seabees, McCullom treated heart and chest patients evacuated from the campaigns across the South Pacific: Guadalcanal, Iwo Jima, Truk, Okinawa.

Vivian and Wiley McCollum show off a favorite Kachina doll in their living room.

During the campaigns from August 1942 to August 1945, the 204th worked steadily, usually overwhelmed with casualties, until the hospital staff were nearly out on their feet. McCollum recalls one stretch when he admitted and treated patients for 45 straight hours.

The interval between campaigns was as dull as the campaign action was intense, and to fill time McCollum organized more baseball games, though the team had dwindled to nine players. By necessity, McCollum became a player/manager; he remembers once having to back-peddle into the Pacific Ocean to haul in one long fly ball.

The last ball game was played in August 1945. Anchored in the bay was a ship loaded with all the gear belonging to the 204th. Its destination was Yokohama, where the invasion of Japan, coded Operation Cornet, would begin. "We hadn't heard about the 1 million estimated casualties of the proposed invasion,

"We got the news that the war had ended from the guy in the radio shack," McCollum says. The relief of not having to shove off for Yokohama, but for home, was immense. "We didn't eat our lunch that day, we drank it."

but we all knew about the Japs' tenacity and willingness to die for the Emperor," McCollum says. "A few days before, I had flown over a little island known as Tinian and noticed that a large part of it was cordoned off. I asked what was going on but no one knew.

"We got the news that the war had ended from the guy in the radio shack," McCollum says. The relief of not having to shove off for Yokohama, but for home, was immense. "We didn't eat our lunch that day, we drank it."

Suddenly, it was all right to think about a future.

* * *

Instead of keeping his heart attack patients in bed for weeks, McCollum was getting them back on their feet, even having them doing moderate exercises.... As might be expected, such innovations were not applauded by his colleagues...

THE DAY BEFORE McCollum shipped out for home, his chief of medicine, Dr. Grey Carter, asked him what he planned to do. McCollum mentioned an internal medicine residency at the Mayo Clinic. His goal was to practice internal medicine and cardiology. Carter, who had been a Rhoades Scholar and a professor of medicine at Yale, said, "You've gotten plenty of internal medicine experience the last few years. What you need is more training in pathology."

McCollum found it accidentally in his own backyard, at the OU medical school, through a fellowship in systemic pathology under Dr. Howard Hopps. There he studied the diseases he had treated and would be treating from the inside out. By correlating the patient's history, including the hospital record, with postmortem exams, McCollum greatly increased his depth of knowledge of disease, disease processes, and anatomical anomalies, particularly of the heart.

Simultaneously, he spent two hours a day with one of the nation's masters of electrocardiography, Robert H. Bayley. Bayley was recognized as such because he and Dr. Frank Wilson had developed an electrocardiograph in the 1930s at the University of Michigan that had set the national standard. Bayley had been working on his own and with engineers ever since to develop an ECG that would record the exact electrical potential of the heart.

By the late 1940s, Bayley was getting pretty close. When he was joined by McCollum, Bayley was working on a three-lead ECG that was two feet tall, four feet long, and a foot deep, and McCollum says it was a wonderfully sensitive tool for Bayley and, gradually, for himself. With Bayley's endorsement, the ECG would be an important component of a new diagnostic heart center at University Hospital, an idea McCollum was trying to sell to department heads at the medical school. Toward the end of his fellowship, McCollum had a state-of-the-art ECG machine. It was contained in a mahogany box that he carried around with him for demonstrations. He took his "magic box," as he called it, to University Hospital, St. Anthony Hospital, and Oklahoma City General. No one was interested in even the concept of a diagnostic heart center.

He wouldn't give up on the idea, but by 1947 his fellowship was over and he and Myrldean had a son, John Scott. It was time to earn a living. He established his private practice in Oklahoma City in July 1947 and for the first six months grossed $85. The McCollums lived on the money he had saved from the service and from the sale of wheat on the land (in the Oklahoma panhandle) they had bought during the war.

Golf, not cardiology, is McCollum's game these days. His backswing gets a workout at least three times a week.

At this melancholy juncture in his young career, McCollum got a break. Dr. Bob Adams asked him to see his wife, who had hypertension. The two doctors barely knew one another, though McCollum had heard that Adams had a substantial industrial practice. Later, Adams showed his gratitude by referring all of his cardiovascular patients to McCollum. Within a few weeks, McCollum never had to worry about patients again.

Adams had asked McCollum to see his wife because he was acquiring a reputation for innovation, even boldness, in his treatment of

cardiovascular diseases. In 1947, five of his patients who had suffered heart attacks subsequently died. Shaken by a feeling of helplessness, McCollum began to put all his heart attack patients on blood thinners. And instead of keeping his heart attack patients in bed for weeks, McCollum was getting them back on their feet, even having them doing moderate exercises to help prevent lethal blood clots from forming. As might be expected, such innovations were not applauded by his colleagues, who more often than not followed the conventional wisdom of the day.

After a golf pro had become disabled with angina, McCollum sent him out on the course with a bottle of nitroglycerine and instructions to take a pill whenever necessary. The pro might take 12 pills a round but that was infinitely better for him, both doctor and patient agreed, than becoming an invalid. The man lived a relatively active life with nitroglycerine for several years, but died of the inevitable heart attack just a few years before the advent of open-heart surgery.

BY THE EARLY 1950s, McCollum was stumping again for a heart center. He estimated it would take about $100,000 to buy the needed catheterization and x-ray equipment. To get ready, he had spent a long, intense week at Johns Hopkins with the world's foremost authority on congenital cardiology, Helen Taussig. After that course, McCollum began traveling throughout the state and region lecturing at county medical society meetings on congenital cardiac abnormalities, and the referrals and interest quickly increased. Often, he would take along his "magic box" to emphasize its high tech and utilitarian appeal.

The heart center didn't materialize, however, until Burt Loy, who had run Oklahoma City General Hospital, the forerunner of Mercy Hospital, had the vision to recognize its potential usefulness, and until a patient of McCollum's, Mrs. Nellie B. Hill, died in 1956. McCollum successfully solicited a donation from her heirs for a heart center at Mercy. The contribution from her estate set the fund raising in motion and within a year the $100,000 had been collected.

Among the donors was 9-year-old Scott McCollum, who received a thank-you note from Mercy's administrator, Sister Mary Coletta: "We are grateful that God makes little boys like you who are interested in helping the doctors and the hospitals to do things for sick people. We hope that you will follow in the footsteps of your daddy, who is an outstanding example of selflessness in devoting his time, energy, and professional ability to helping others."

In his memorabilia room, filled with items such as pictures of his prize Limousin bulls and gifts from former patients, McCollum demonstrates a model of the original stethoscope.

The center's staff included McCollum, as the first and only board certified cardiologist in Oklahoma, cardiovascular surgeons Allen Greer, John Carey, Nahzi Zuhdi, and inventor Garman Kimmell of the Kim-Ray Corporation, whose glucose prime pump saved blood by recirculating it. Now all the pieces were in place to diagnose and correct life-threatening or crippling cardiac abnormalities in babies and children. By 1969, the team was doing the state's first heart by-pass surgeries.

One of the early candidates might have been Dr. Bayley, a paradigm of the Type A personality, relentlessly driven to achieve perfection and while he was at it, smoking four to five packs of cigarettes a day. Despite McCollum's efforts to get his friend to quit, Bayley wouldn't change a thing because he believed any change of behavior would deleteriously affect his work and he was getting very close to developing an ECG that would precisely reflect the heart's electrical potentials. Unfortunately, Bayley died in 1969 at the age of 64.

Although McCollum had quit smoking for good in 1968, he had already been on the

"Wiley could be inflexible and obstinate. That's a side many of his colleagues saw. But the thing that used to make 'em so damned mad was that his way so often was the right way."

—A.C. Lisle, MD

receiving end of several surgeries himself. Yet, he always bounced back strongly. Myrldean's health, on the other hand, had never been never robust, and by 1974 she had become fragile. She broke a hip in December 1977. A few days later, their son Scott, 30, was killed when the pick-up truck he was driving on the family's ranch near Lawton skidded on an icy road and rolled over four times.

According to one of McCollum's best friends, neurosurgeon A.C. Lisle, McCollum's salvation was his work. While McCollum agrees, the time between calamities was short. Later that year, Myrldean developed the first signs of ovarian cancer. "She had wanted me to retire and so in the spring of 1981 I sort of semi-retired and bought a place in Rancho Bernardo, California," McCollum says. But it was too late for Myrldean, who died in November. McCollum spent most of his time there as a widower.

He remarried a year later, but it never took. As Dr. Lisle says, "Wiley's second wife once told me, 'He's a remarkable man. He'll give you the shirt off his back and then in five minutes, he'll tear it off you.' I think she had it right. While he is very compassionate, especially toward his patients, Wiley could be inflexible and obstinate. That's a side many of his colleagues saw. But the thing that used to make 'em so damned mad was that his way so often was the right way."

* * *

WILEY MCCOLLUM WOKE UP, literally and figuratively, on July 4, 1984, and at that moment he decided to retire. In the decade since, he's gotten divorced, gotten out of the Limousin cattle business, and sold off his ranch and farmland. The land he had donated in 1983 to his alma mater, Oklahoma State, for a new golf course became larded down in politics for almost a decade, and it was only in 1994 that the course was dedicated. Summing it all up, McCollum advises, "Never donate anything to the government."

On the plus side, McCollum, in 1991, married Vivian, his former office manager from the late Sixties and early Seventies. They are together now, Vivian happily assisting Wiley with his business affairs. He would "fill her in" if the phone didn't keep ringing and the visitor didn't keep asking questions. Even so, they find time to travel and play golf together.

"Medical students used to ask me, 'Dr. McCollum, what was the most important part of your training?' My answer just dumbfounded them. I said, 'It was the years I worked in a grocery store in Waynoka.'

"'Huh?' they'd say. So I'd spell it out.

"'That's where I learned how to communicate with people. If you can't communicate, it doesn't matter what you know. That's just common horse sense.'"

Begun in 1981, the Leaders in Medicine series has recognized some of Oklahoma's most outstanding physicians. This is the twenty-sixth biography in the series.

Richard Green is a free-lance writer based in Oklahoma City and has been writing the Leaders in Medicine stories for a number of years.

Victor R. Rivas, a Norman-based photographer, returns to the Leaders in Medicine series after an absence of several years.

Quo Vadis?

Philip J. Maguire, MD

There is an explosion of DNA and genetic research today. The dignity of life dictates caution be urged in the use of preembryos and fetal parts for these studies.

As youngsters we were frightened and horrified by Frankenstein. That wonderful story written by Mary Shelley has become a legend. Frankenstein is now part of our vernacular representing almost any monster and even monstrous events—the word itself evoking badness personified.

In truth Dr. Frankenstein's goal was not to create something evil but to become the first transplant surgeon. He was really trying to create life. It was his assistant who picked up the wrong brain. Therein lies a threat we face in medicine today—someone picking up the wrong brain.

The good doctor's work with cadavers has always been treated as sort of a joke. But maybe not anymore. Enter embryo research. Today scientists want to and are able to use fetal parts to grow new people! Well, not quite yet, but they're getting close. Embryos can be duplicated in the laboratory. The ability to engineer the genes of individual cells and alter their function is astonishing. Whether this will lead to a modern day Frankenstein remains to be seen.

It is possible to remove several eggs from an ovary and fertilize them in a dish. One fertilized egg is returned to the mother's uterus and the remaining are frozen as "back-ups." A single sperm cell can be placed in any appropriately ripe egg. This is old stuff today. Now the embryos themselves can be duplicated. Cloning is here!

Preimplantation studies of DNA gene sequence and gene therapy are aspects of embryo research that offer tremendous possibilities for disease control and prolonging life. But the use of eggs from aborted fetuses—are they a source for new life or is it something artificial? There is a root problem about origins and parenthood and family and what constitutes these things. Because it *can* be done doesn't always dictate that it should be

Direct correspondence to Philip J. Maguire, MD, 1124 N.W. 50th Street, Oklahoma City, OK 73118.

done. The ethical nature of the question stems from the possibility of several choices, all equally possible.

For those who believe that embryos have the same rights as any other human being, these events are troubling. A surgeon at Massachusetts General Hospital stated that study groups "should not allow events or public sensitivities to limit our vision as to what the potential values of this type of research would be." One has to wonder who, then, is supposed to influence researchers. Should any checks and balances be proscribed?

Pig litters have been altered genetically so that their organs, normally rejected by humans, could be transplanted into humans.

OF COURSE there are many wonderful aspects of this research. One young woman whose liver made excess cholesterol recently had part of her liver removed. Virus cells with the missing gene were injected into the liver cells and returned to her. The results were successful, and she had a significant reduction in cholesterol. Pig litters have been altered genetically so that their organs, normally rejected by humans, could be transplanted into humans.

Preembryo gametes up to fourteen days (before development of the primitive streak) are vaguely regarded as not yet human. But from preembryos up to eight-cell stage, blastomeres, single cells can be removed and the remainder can still produce a complete adult. The single blastomere can develop into complete individuals, and cells from two different preembryos can be combined to form a chimera.

The problem might arise trying to figure out who is who. Ms. A donates an egg; Mr. B donates a sperm. The embryo is implanted into the uterus of Ms. C who carries the child for Mr. and Mrs. D. In Italy a "father" was allowed to legally disown a son born to his spouse by donor sperm. Similar widely publicized cases here seem outrageous but they are real.

In vitro fertilization is also certainly providing unbelievable new information regarding human development and disease. As scientific skill becomes more sophisticated, it appears there is no end to how far we can delve into life. Is it possible we'll find the fountain of youth through the use of fetal cells and recombinant DNA? Hope springs eternal for life everlasting!

Since human embryos have been cloned, exact duplicates of children are possible. If one knew in advance the kid was going to be a little brat, it could be deleted. Pretty little girls—order two. One could clone an embryo, save the extra, and if the kid got run over by a car, go ahead and develop the spare. Will we have an engineered superperson? For cultures that desire male infants—no problem; presex the embryos. So babies don't come just from a loving relationship but can be ordered to size and appearance.

Or a large number could be cloned for women who can't conceive, but then there would be about 30 little kids who are identical twins. Of course, other genes could be inserted into each one later to mix them up somewhat.

How far we have come since April 1953 when Drs. Watson and Crick first outlined the structure of DNA. As scientists we are always interested in what is next. Yet there must be some dignity to life even for embryos. Using the old ethical premise of the "law of twofold effect," can we destroy an embryo to use its cells for a greater good?

As we approach the great mystery of life itself, the answers are not easily forthcoming, but the question must be asked—"Quo Vadis?"

The Author
Dr. Philip J. Maguire practiced obstetrics and gynecology in Oklahoma City for 28 years and is now doing volunteer medicine for free clinics.

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Board of Trustees tackles a variety of topics at summer meeting

(Left) Dr. Bertha M. Levy, Oklahoma City, receives the Wyeth-Ayerst Physician Award for Community Service from Dr. Walter E. Brown. Tulsa. (Right) Doris Pitts and her family thank the board for its special recognition of her late husband, Dr. James B. Pitts, MD, Oklahoma City.

AWARDS headed the agenda at the August 21 meeting of the OSMA Board of Trustees meeting in Oklahoma City.

A special memorial resolution was presented to Doris Pitts in memory of her late husband and OSMA past president James B. Pitts, MD. Dr. Pitts practiced obstetrics and gynecology in Oklahoma City for many years and was very active in organized medicine.

Dr. Bertha M. Levy, named recipient of the Wyeth-Ayerst Physician Award for Community Service at the OSMA's Annual Meeting in April, was present to receive a handsome commemorative plaque and the appreciation of her colleagues. A retired Oklahoma City pediatrician, Dr. Levy also spent a number of years administering the state's Medicare and Medicaid programs. She was featured in a Leaders in Medicine biography in the December 1993 JOURNAL.

In other business, President Jay A. Gregory, MD, reported that Southwestern Bell Telephone is giving grants for a project in telemedicine in a rural setting. While costs are still too high for the OSMA to become involved in the project, Dr. Gregory has been named as a judge to help choose grant recipients.

Secretary-Treasurer Carol Blackwell Imes, MD, delivered a comprehensive report in accordance with the recommendations of the Financial Oversight Committee and thanked OSMA Comptroller Shirley Burnett for her expertise and assistance in preparing the internally generated report. Dr. Imes noted that the OSMA currently is over budget because of money given to assist the start-up of the new MCO, PROklahoma, but the funds are expected to be recovered in the coming months (a line of credit with a local bank also has been established to help fund the MCO). Normally the association keeps some $600,000 is reserves. Dr. Imes also reported that the accounting firm of Arthur Andersen has been selected to perform the extensive internal audit approved earlier this year.

OSMA Past President G. Lance Miller, MD, reported that work on the organization of PROklahoma is proceeding and a prospectus is being prepared. It is hoped the entire prospectus package can be delivered to physicians by the first of October. Dr. Gregory and OSMA Executive Director David Bickham will be attending county society meetings across the state to educate physicians about the new MCO, and trustees were encouraged to do the same at their hospital meetings. In addition, an educational videotape about PROklahoma is being readied for distribution.

Mike Sulzycki, OSMA associate director and director of communications, reported on Dr. Perry A. Lambird's campaign for AMA vice-speaker. Copies of a "bare-bones" campaign budget were distributed and plans for activities at the AMA Interim Meeting in December were discussed. Dr. Lambird said his plans include an "afterglow" at the meeting. Dr. Mary Anne McCaffree, speaker of

And that was the weekend that was ... August 19-21, 1994

IT WAS a hot August weekend, but things didn't slow down at the Oklahoma State Medical Association.

Friday, the 19th, a select group of OSMA leaders met with American Medical Association consultants in Oklahoma City as part of the AMA's consortium "Study of the Federation," an ongoing process designed to envision the future of the federation and how it must be structured to meet the rapidly changing needs of today's and tomorrow's health care professionals and their patients. Oklahoma was one of only six states selected to participate in intense, state-level consortium meetings with AMA consultants. The state focus study group was attended by OSMA President Jay A. Gregory, MD, as well as Drs. Deborah L. Huff, M. DeWayne Andrews, John L. Aldridge, Philip Mosca, Ray V. McIntyre, and Gary L. Paddack. Ms. Cate Bower, consultant to the AMA, led the discussion, and OSMA Associate Director Mike Sulzycki was present as staff liaison.

At the AMA's consortium study were (l to r) Cate Bower, AMA consultant, and Drs. John Aldridge, Deborah Huff, Philip Mosca, Gary Paddack, Jay Gregory, and Ray McIntyre. Dr. DeWayne Andrews, who also attended, had to leave early.

The day ended on a lighter note—with a splash, if you will—at the annual OSMA Student Picnic in the Oklahoma City Zoo's Aquaticus pavilion. First-year medical students and their families gathered with a number of OSMA officers and staff members for a last look at summer and a first look at organized medicine before settling into their busy fall routines. Some 400 individuals of all ages were present for a good old-fashioned picnic of hamburgers, hot dogs, soft drinks, and beer in the Aquaticus grandstand. The evening of fun was capped with a show by the Aquaticus dolphins.

In the Oklahoma City Zoo's Aquaticus pavilion, medical students, their families, and guests serve themselves at the annual OSMA Student Picnic.

Saturday the mood grew serious again as Oklahoma's county medical society and specialty society presidents met at OSMA headquarters for a leadership briefing. OSMA Executive Director David Bickham explained in detail the organizational structure and general operations of the OSMA. Dr. Gregory followed with a description of the medical federation—from county societies, up through the state associations, to the AMA at the national level. Present from the AMA were Susan Raef and Keith Pierson, who described what the AMA can and does do for its members and what counties societies can do on a local level to support and encourage organized medicine.

That evening, many of those attending the briefing joined OSMA trustees and their spouses at the Cowboy Hall of Fame for a dinner hosted by C.L. Frates and Company. Guests were the first to see a draft of a new video that will explain PROklahoma to groups of physicians across the state.

Sunday the OSMA Board of Trustees convened for their quarterly August meeting and worked through a varied agenda. [A report of that meeting begins on page 471.]

Board of Trustees *(cont.)*

the OSMA House of Delegates, suggested sending a solicitation letter to OSMA members and Mr. Bickham pointed out that the OSMA budget already includes funding for such campaigns.

Trustees were urged by Dr. William O. Coleman to attend the AMA Interim Meeting as representatives of their hospital medical staffs.

Mr. Bickham reported that an agreement has been drawn up for the Mark R. Johnson Memorial and encouraged county medical societies to make contributions to the trust. The trust will fund a competition and award for "Excellence in Medical Writing" for medical students and residents. [Announcement of the competition by the Board of Trustees and the Editorial Board appears on page 473.]

Mr. Bickham also reported that the OSMA Committee on Family Violence had produced an excellent booklet, and that he was seeking financial support from various organizations for its publication. Dr. Edward N. Brandt, Jr., chairman of the Committee on Family Violence, said copies of the informative booklet will go to all OSMA members. The board then approved a motion to spend up to $4,000 for the booklet's publication.

Life Memberships in the OSMA were approved for Frederick C. Wallingford, MD, Bartlesville, and Orange M. Welborn, MD, Ada.

Dr. Brandt, speaking as chairman of the American Cancer Society Legislative Committee, urged support of State Question 667, which would establish a $1 entertainment tax to fund breast cancer research and mammograms for indigent women. The board approved a motion that would provide for the publication of an educational article on the question in the *OSMA News*, make educational material for patients available to physicians, and endorse support for the question by the OSMA Council on Public and Mental Health.

Greatest concern: career politicians

Muskogee's Coburn wins nomination for Second District seat

THOMAS A. COBURN, MD, a family practitioner in Muskogee, breezed through the August primary to win the Republican nomination for Oklahoma's Second District seat—the seat currently held by seven-term Democrat Mike Synar.

Dr. Tom Coburn

Coburn has been labeled a "dynamite candidate" by L. William Paxon, a New York Republican who heads the party's committee dedicated to electing members to the House of Representatives.

A professed conservative, Coburn says he favors a balanced budget, the line item veto, reduced federal spending, term limits, and strong anti-crime legislation. He opposes gun control.

Regarding health system reform, he says President Clinton's plan "would drive up taxes and costs, reduce the options enjoyed by the average American, and place a crushing burden on small business. Instead we should focus on insurance reforms, medical savings accounts, and subsidies for the truly needy."

Coburn says his greatest concern is the emergence of a professional political elite epitomized by career politicians.

"More and more we are represented in Congress by politicians... who do not live in our communities, who do not share our values, who do not understand our problems, and who do not respond to our needs and wishes."

A successful businessman for eight years before deciding to enter medicine, Coburn was president of his class at the University of Oklahoma College of Medicine, where he was graduated in 1983. After completing his postgraduate training he returned to Muskogee, his childhood home, to practice.

Coburn is married to his high school sweetheart, the former Carolyn Denton, and they have three daughters.

Those individuals wishing to support Dr. Coburn's campaign can reach the Tom Coburn for Congress Committee at (918) 686-9495 or P.O. Box 504, Muskogee, OK 74402.

Announcing
The Mark R. Johnson Competition
"Excellence in Medical Writing"

The Editorial Board of the JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION and the OSMA Board of Trustees are proud to announce the Mark R. Johnson Competition—Excellence in Medical Writing.

A $500 cash award will be presented to the medical student or resident at the University of Oklahoma College of Medicine (Oklahoma City or Tulsa campus) who, by February 1, 1995, submits the most worthy scientific paper or opinion piece for publication in the JOURNAL.

Entries will be judged by the JOURNAL's Editorial Board at its annual meeting in March, and the winner, if any, will be announced at the Annual Meeting of the OSMA House of Delegates in April. All decisions of the Editorial Board will be final.

The student or resident submitting the paper need not be the *sole* author, but must be the *lead* author and must have done the majority of the writing. Entries in the competition should be clearly labeled as such when they are submitted.

The memorial trust that funds the competition was established by the friends and family of Mark R. Johnson, MD, who, during his two decades as editor-in-chief of the OSMA JOURNAL, exemplified the very best in both expository and opinion writing in the field of medicine.

Break the silence, end the violence

Lines of Defense: Domestic Violence Awareness Month

By Robert E. McAfee, MD
President
American Medical Association

AMERICA is a nation at war, and the struggle is as close as our own living rooms. Every day we read about gangs battling on our urban streets, the pernicious hold drugs still have on many of our youth, and the continuing fight against AIDS. We are less likely, however, to read about the war played out within the American family, where the front lines lie hidden behind the front doors of our communities. This is the battlefield of domestic violence, where the casualties are enormous and the heroes far too few.

It has been said that heroes are those who do what they can. With so much that physicians can do in this crisis, the hour for quiet heroism is upon us. October is Domestic Violence Awareness Month, a chance for us to take stock of our efforts. As we do, however, we must remember that for the victims, domestic violence is not merely a month-long affair—it is a daily, a weekly, and all too often, a lifelong, struggle.

Robt. McAfee, MD
President, AMA

By the time you have finished reading this sentence, another woman in the United States will have been beaten, battered, and bruised by someone she knows. Every 12 seconds, 24 hours a day, 365 days a year, a woman is punched, kicked, slapped—or worse. Every decade, domestic violence kills as many women as the total number of Americans who died in the Vietnam War.

The damage does not stop, however, with the lives and bodies of women. Domestic violence targets all of the most vulnerable among us, including small children and the frail elderly. Two thousand American children are beaten and starved to death every year, and millions more suffer the horrific effects of ongoing abuse and neglect. It is hard to measure how many millions more victims are ignored or overlooked, *because physicians fail to properly identify as many as 95 percent of the victims of domestic violence.*

The security of a physician's office may be the only sanctuary the patient can find in a personal hell of violence and neglect. We have a responsibility—not only as physicians, but as citizens

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and community members—to create the necessary impact to stem this growing American epidemic.

We must begin with our own awareness of violence in the family. I will never forget the day I met a cardiologist who started a hospital-based treatment center in Kenosha, Wisconsin, for abused women.

"I understand you treat battered women," I said as we were introduced.

He replied, "And so do you." My education began at that instant.

What he meant was that when physicians examine patients with cuts and bruises and broken arms, we may actually be treating victims of abuse.

With a problem as prevalent as domestic violence, the AMA's guidelines for diagnosis and treatment recommend *routine* screening in the primary care setting. We must be on the lookout, because AMA research shows that 80 percent of Americans feel they could tell a physician if they had been either a victim or a perpetrator of family violence. Patients trust their physicians. What physicians need are the right tools, and the AMA has them at hand.

In 1990, we formed the National Coalition of Physicians Against Family Violence, now more than 6,000 members strong. The Coalition keeps physicians informed about resources, model programs, speakers, and public education materials. Participants receive the AMA Diagnostic and Treatment Guidelines on child and elder abuse and domestic violence, as well as patient materials bearing the message, "When you break the silence, you begin the cure."

It will take more than openness or good medicine to make the cure complete, however. Physicians should become familiar with the range of resources in the community: shelters, legal advocacy, counseling, support groups, crisis hotlines, and your state Alliance. Look also to the extraordinary grass roots efforts of our own Federation, where physicians like you have already taken this issue to heart. To turn the tide in a war of this magnitude is going to take every available recruit, side by side, attacking on as many fronts as possible. It is going to take lawyers, judges, and the police, concerned citizens and local communities; all of us engaged in health care; and the victims themselves—all working together.

"I understand you treat battered women," I said... He replied, "And so do you."

To do *your* share to recognize and treat victims of domestic violence, just keep your professional RADAR turned on:

- **R**emember to screen your patients about violence.
- **A**sk questions such as: "At any time, has a partner hit, kicked, or otherwise hurt or frightened you?"
- **D**ocument your findings. Information in the patient's chart about "suspected domestic violence" can serve a valuable function in court should the patient seek legal action. A physician's documentation validates the victim's allegations.
- **A**ssess your patient's safety. Is it safe to go home? Find out if any weapons are kept in the house, if the children are in danger, and if violence is escalating.
- **R**eview options with your patient. Know about referral options.

Remember, the public entrusts us with their care, and many of us have already chosen to be the heroes that their physical and emotional pain demand. But to gain a beachhead against this national epidemic, there is not a person—or a moment— to spare. As physicians, as citizens, as parents, as sons and daughters, we must work together to break the silence and end the violence, beginning now with Domestic Violence Awareness Month, and throughout this and every year.

Note: A 40-page booklet on family violence, produced by the OSMA Committee on Family Violence, is being printed and copies will be distributed to all OSMA members. It includes valuable guidelines for identifying domestic violence, interviewing the patient, and documenting findings, as well as a discussion of legal considerations. There is also a comprehensive list of domestic violence programs and shelters in Oklahoma and information about joining the AMA's National Coalition of Physicians Against Family Violence.

It's your association

Trustees conduct association's business on behalf of members

IN AUGUST the JOURNAL began a series of informative articles about the organization and operation of the OSMA. Last month's article dealt with the election, terms of office, and duties of OSMA's leaders, including the Board of Trustees. Space did not permit the inclusion of Chapter V of the OSMA bylaws (revised 1993) which deals with the Board of Trustees, so it appears below.

Currently Dr. Douglas C. Hubner, Tulsa pathologist, is chairman of the board and Dr. Chester L. Bynum, Norman radiologist, is vice-chairman.

Effective at the end of April's elections in the House of Delegates, OSMA's trustees and alternate trustees for 1994-95, including the last year of their current term in office, are as follows:

District I
Trustee: Richard E. Martin, MD, Pryor, 1997
Alternate: Douglas G. Cox, MD, Grove, 1997

District II
Trustee: Robert Phillips, MD, Stillwater, 1997
Alternate: Mark Palmer, MD, Ponca City, 1997

District III
Trustee: David Selby, MD, Enid, 1997
Alternate: James S. Gerber, MD, Okarche, 1997

Bylaws *(continued)*

District IV
Trustee: William T. Morris, MD, Woodward, 1997
Alternate: Ed L. Calhoon, MD, Beaver, 1997

District V
Trustee: Frank K. Buster, MD, Cheyenne, 1997
Alternate: James R. Rhymer, MD, Clinton, 1997

District VI
Trustees: Jon C. Axton, MD, 1995; Donald R. Carter, MD, 1995; and Clarence Robison, MD, 1996; Oklahoma City
Alternates: M. DeWayne Andrews, MD, 1995; Wayne Wasemiller, MD, 1995, and Royice Everett, MD, 1996; Oklahoma City

District VII
Trustee: Chester Bynum, MD, Norman, 1995
Alternate: David Holland, MD, Shawnee, 1995

District VIII
Trustees: Boyd O. Whitlock, MD, 1995; Douglas C. Hubner, MD, 1995; and W. Frank Phelps, MD, 1995; Tulsa
Alternates: C. Wallace Hooser, MD, 1995; David Harper, MD, 1995; and David Confer, MD, 1995; Tulsa

District IX
Trustee: Fred Ruefer, MD, Muskogee, 1997
Alternate: Bonnie Ashing, MD, Tahlequah, 1997

District X
Trustee: R. Kern Jackson, MD, McAlester, 1995
Alternate: Charles Cook, MD, Poteau, 1995

District XI
Trustee: Robert Hayes, MD, Atoka, 1996
Alternate: Jone Kendrick, MD, Idabel, 1996

District XII
Trustee: Gary L. Paddack, MD, Ada, 1996
Alternate: James V. Miller, MD, Ardmore, 1996

District XIII
Trustee: Robert J. Weedn, MD, Duncan, 1997
Alternate: Richard J. Boatsman, MD, Lawton, 1997

District XIV
Trustee: Jeffrey Lester, MD, Mangum, 1997
Alternate: Joseph Krueger, MD, Altus, 1997

Copies of the complete OSMA bylaws are available to any member upon request.

Chapter V
Board of Trustees

Section 1.00 Composition. The Board of Trustees is composed of the Association's general officers and Trustees to be elected from each of the fourteen authorized Trustee Districts. Each district shall be entitled to one Trustee for every 500 physician-members or fraction thereof. In computing the physician population of each Trustee District, only Active, and Active Limited-Dues members shall be counted. An Alternate Trustee shall be elected for each Trustee, but such Alternate Trustee shall serve only in the absence of the Trustee. The Chair of the Board of Trustees may invite such ex-officio members to meetings as deemed necessary or advisable in the conduct of business.

Section 2.00 Election of District Trustees. Trustees and Alternate Trustees shall be elected by the House of Delegates at the annual meeting, based upon nominations made by the Delegates from their respective districts. In Trustee Districts consisting of more than one county, the Trustee and Alternate Trustee shall not be elected from the same county. (Only Active members may be nominated and/or elected to a Trustee or Alternate Trustee office. See Chapter I, Section 2.01).

2.01 Determination of Representation. The determination of the number of Trustees to be elected from each of the fourteen authorized Trustee Districts shall be had by computing the number of Active and Active Limited-Dues members residing in each Trustee District on December 31 of the year preceding the annual meeting at which the Delegates elect the Trustees from the various Trustee Districts.

Section 3.00 Terms of Office. All Trustee and Alternate Trustees representing Trustee Districts shall be elected at the annual meeting for concurrent terms of three years each. The general officers shall serve on the Board of Trustees during their terms of office.

3.01 Tenure. No District Trustee shall be elected for more than two full consecutive terms, and shall be ineligible at the expiration of that term for immediate election to Alternate Trustee. This limitation does not apply to Alternate Trustees.

Section 4.00 Apportionment of Trustee Districts. The State of Oklahoma has been divided into fourteen (14) Trustee Districts, as provided in Chapter IV, Section 7.00, composed of the following counties:

District No. 1 - Craig, Delaware, Mayes, Nowata, Ottawa, Rogers, Washington.
District No. 2 - Kay, Noble, Osage, Payne, Pawnee.
District No. 3 - Alfalfa, Garfield, Grant, Kingfisher, Logan, Major, Woods.
District No. 4 - Beaver, Cimarron, Dewey, Ellis, Harper, Texas, Woodward.
District No. 5 - Beckham, Blaine, Canadian, Custer, Roger Mills.
District No. 6 - Oklahoma.
District No. 7 - Cleveland, Creek, Lincoln, Okfuskee, Pottawatomie, McClain.
District No. 8 - Tulsa.
District No. 9 - Adair, Cherokee, McIntosh, Muskogee, Okmulgee, Sequoyah, Wagoner.
District No. 10 - Haskell, Hughes, Latimer, LeFlore, Pittsburg, Seminole.
District No. 11 - Atoka, Bryan, Choctaw, Coal, McCurtain, Pushmataha.
District No. 12 - Carter, Garvin, Johnston, Love, Marshall, Murray, Pontotoc.
District No. 13 - Caddo, Comanche, Cotton, Grady, Jefferson, Stephens, Tillman.
District No. 14 - Greer, Harmon, Jackson, Kiowa, Washita.

4.01 Change of Districts. The House of Delegates may revise the composition of Trustee Districts. Such revision shall require a two-thirds vote of the Delegates present.

Section 5.00 Meetings. The Board of Trustees shall meet as provided by the Constitution. The Annual Meeting of the Board shall be held immediately prior to the Annual Meeting of the Association House of Delegates. The Board may also meet by telephone conference call, closed circuit television, or by any other electronic means that would allow all Board members to interact.

5.01 Quorum. At any meeting of the Board of Trustees, a majority of the Trustees or their alternates shall constitute a quorum.

5.02 Presiding Officer. The presiding officer of the Board of Trustees shall be the Chair of the Board, who is elected by the Board from among its own members at its annual meeting. The term of office of the Chair is one year, and he/she is limited to three consecutive terms in this position. A Vice-Chair shall be elected by the Board from among its own members to serve concurrently with the Chair and according to the same limitation of tenure. The Vice-Chair shall preside in the absence of the Chair of the Board, or at the discretion of the Chair. Neither the Chair nor the Vice-Chair may be elected to a term of office that will extend beyond their tenure as an

elected Trustee. The President of the Association is ineligible for election to either office.

Section 6.00 General Duties. The Board of Trustees shall perform all acts and transact all business for or on behalf of the Association, and shall manage the finances, property, and conduct the affairs, work and activities of the Association, except as may be otherwise provided in the Constitution or Bylaws. It shall carry out or supervise the attainment of directives from the House of Delegates. When the House of Delegates is not in session, the Board of Trustees may exercise legislative powers normally conferred upon the House, provided that no action may be taken which is contrary to a general or specific policy prescribed by the House of Delegates.

Section 7.00 Specific Duties. In addition to the rights and duties conferred or imposed on the Board of Trustees elsewhere in the Constitution and Bylaws, the Board of Trustees shall perform other duties as prescribed in this section.

7.01 Annual Report. The Board of Trustees shall report annually to the House of Delegates at the time of the annual meeting. The report shall be presented on behalf of the Board of Trustees by the Chair of the Board.

7.02 Budget and Audit. The Board of Trustees shall prepare an annual budget of income and expenses, and shall present it to the House of Delegates for consideration. The report shall be presented for the Board of Trustees by the Secretary-Treasurer. The Board of Trustees may prescribe a method of accounting for the Association.

7.03 Judicial Authority. The judicial power of the Association shall be vested in the Board of Trustees, whose decision on behalf of this Association shall be final.

7.031 Original Jurisdiction. The Board of Trustees shall have original jurisdiction in: (a) All questions involving the interpretation of the Constitution and Bylaws of this Association; (b) All controversies involving ethics or arising under this Constitution and Bylaws to which the Oklahoma State Medical Association is a party; (c) All controversies between two or more component societies, or between a component society and a member of another component society; (d) All matters where a showing is made that an objective forum cannot be obtained or has not been obtained in a component society, based upon the motion of a member of the Board of Trustees which is supported by a majority of Trustees present; and (e) All matters of professional conduct properly referred to the Board of Trustees by the Committee on Medical Ethics and Competency as set forth in Chapter X (Sections) 4.06 (d) and 4.07 of these Bylaws.

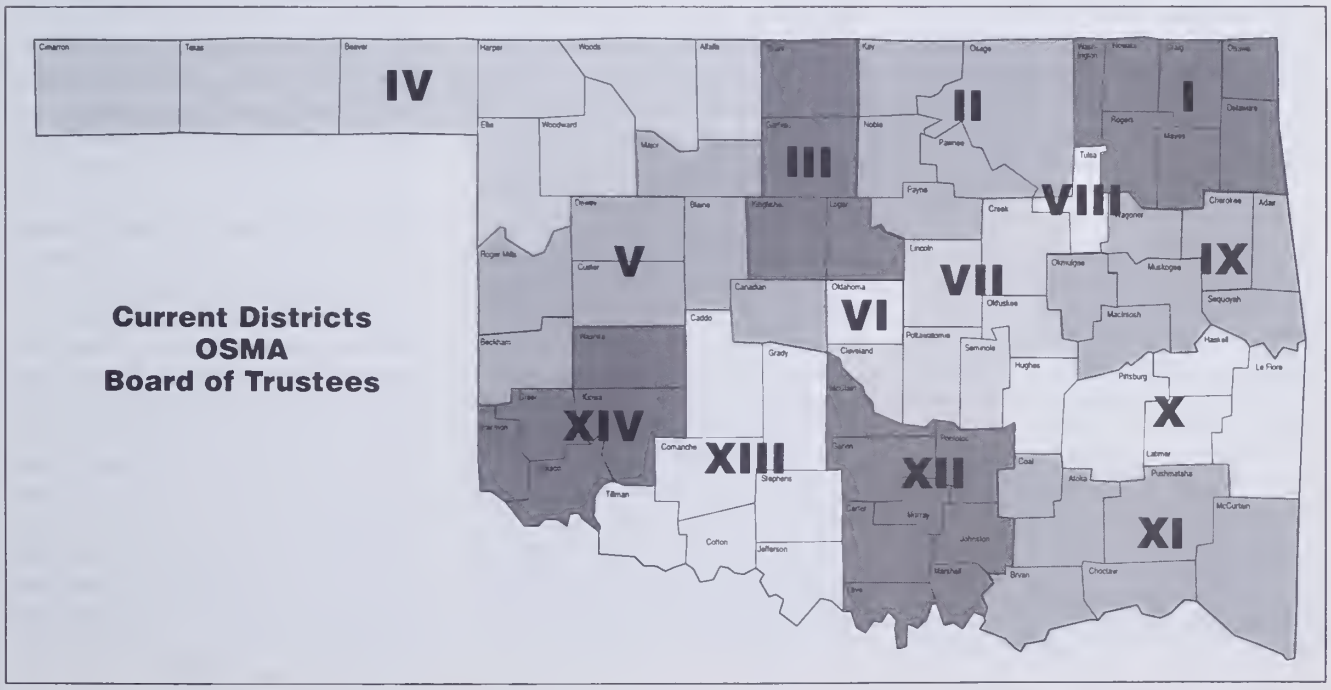
7.032 Delegation of Authority. The Committee on Medical Ethics and Competency of the Association shall be the investigating committee of the Board of Trustees on matters involving judicial decision. The Board of Trustees, in its discretion, may refer appropriate complaints or controversies to the Committee for investigation under the rules governing the Committee's procedure; and it shall receive and act upon matters properly brought to its attention by the Committee on Medical Ethics and Competency. In either instance where the Committee becomes involved in a problem of original jurisdiction, it shall report its findings and recommendations to the President and Chair of the Board, and shall prefer and prosecute any charges before the Board of Trustees.

7.033 Appellate Jurisdiction. The Board of Trustees shall have appellate jurisdiction for this Association in all cases arising between a component society and one or more of its members. Notice of appeal shall be filed with the Board of Trustees in writing within 30 days of the date of the decision by the component society, and the appeal shall be perfected within 60 days from the date of notice.

7.034 Meetings, Rules or Procedure. The Board of Trustees may function in judiciary capacity at any annual or called meeting. It shall enact its own rules of procedure for hearing and disposing of cases of original or appellate jurisdiction; provided, however, that all parties to a controversy, including the Committee on Medical Ethics and Competency and the Board of Trustees, are entitled to legal counsel, adequate notice of charges, adequate notice of hearing, and the right of cross examination.

7.035 Penalties. In carrying out its responsibilities involving problems of original Association jurisdiction, the Board of Trustees may penalize any member of the Association who has been found guilty of unprofessional conduct, ethical or organizational violations by: (a) Official written reprimand to the member and written notice to the officers of his/her component medical society; (b) Suspension from membership for a fixed period of time; (c) Termination of membership; (d) The reporting of gross offenders to the Oklahoma State Board of Medical Licensure and Supervision; or (e) Any appropriate combination of such penalties. When acting in an appellate capacity, the Board of Trustees may affirm, reverse or render such judgment as should have been rendered by a component society.

(continued)



HEALTH DEPARTMENT

Inattentiveness and impulsivity mark attention deficit disorder

Attention deficit disorder (ADD), or attention deficit hyperactivity disorder (ADHD), is a developmental disability estimated to affect between 3% and 5% of all children. Inattention and impulsivity are the most predominant characteristics of the disorder, with hyperactivity occurring in many but not all cases.

The inattentiveness in a child with ADD/ADHD will be seen as difficulty with selective attention (zeroing in on the relevant task), sustaining attention (maintaining attention through distractions), or dividing attention (doing two or more tasks at the same time).

Impulsivity, another common characteristic of the ADD/ADHD child, is displayed when the child often acts without thinking or has great difficulty waiting their turn. Risk-taking behavior results from difficulty controlling impulses in a dangerous situation rather than the deliberate act of taking a risk.

The combination of inattentiveness and impulsivity often cause the child with ADD/ADHD to be quite disorganized. Hyperactivity occurs in most, but not all, ADD children and can range in intensity from mild to severe.

Accurately diagnosing ADD/ADHD involves evaluation by multiple professionals, with multiple techniques, in multiple settings. The elements of an evaluation usually include: (1) a thorough medical and family history, (2) a physical examination, (3)
interviews with the child, parents, and teacher, (4) behavior rating scales, (5) observations of the child, and (6) psychological tests measuring cognition, social and emotional adjustments, and learning disabilities.

Your local county health department guidance centers are available to assist physicians in dealing with ADD/ADHD children, their families, and schools. Contact your local guidance center or your county health department for more information and assistance.

WORTH REPEATING

First Lady answers MD's letter

The June 1994 JOURNAL *reprinted a letter from Dr. S.A. Dean Drooby to Hillary Rodham Clinton. In it, he detailed his concerns about the Clinton health plan. On August 16, Mrs. Clinton responded as follows:*

Dear Dr. Drooby:
Thank you for joining us in Muskogee on April 8. I am sorry that you were not able to share your thoughts on that occasion. Your insightful letter raises some of the issues at the heart of the debate on health care reform.

You note that we must maintain the high quality of healthcare in this country. As I have listened to physicians and other health professionals share their views about the current healthcare system, I have heard that too many Americans are unable to get the health care they need. I have also learned that too many physicians and hospitals are forced to absorb the costs of providing emergency care to uninsured individuals—who are unable to get preventive services and counseling and instead must wait until their illnesses are serious and costly before seeking care. It is clear, however, that we have the highest quality of care, the most talented and dedicated health professionals, and the most advanced research institutions in the world. The bills that Congressman Gephardt and Senator Mitchell have introduced will preserve the exceptional quality of our health care system and ensure that all Americans have insurance coverage and real access to our high quality professionals and institutions.

You also raise concerns about the al-

Bylaws *(continued)*

7.036 Appeals to the American Medical Association. Judicial decisions of the Board of Trustees may be appealed to the Council on Ethical and Judicial Affairs of the American Medical Association in accordance with that organization's Constitution and Bylaws. In such event, the decision of the Board of Trustees shall not be suspended pending the appeal to the AMA Council on Ethical and Judicial Affairs.

7.04 Issuance of Charters. A medical society desiring to become a component society of the Association shall file a request for a charter, together with a copy of its constitution and bylaws, with the Association. Upon approval of the Board of Trustees, and authorization by the House of Delegates, a charter, signed by the President and Secretary-Treasurer of the Association, shall be issued.

7.041 Revocation of Charters. The Board of Trustees may recommend to the House of Delegates the revocation of a charter for cause.

7.05 Amalgamation. The Board of Trustees may approve the amalgamation of two or more component societies, and may approve separations of such societies upon request. Final authorization shall be by action of the House of Delegates.

7.06 Central Office. The Board of Trustees shall establish and maintain a central office.

7.07 Fifty Year Pins. The Board of Trustees may award Fifty Year Pins to members who have practiced medicine for 50 years or more since graduating from medical school. Application for such awards must be initiated by component medical societies and, upon approval, the pins shall be presented to the recipients by appropriate members of the Board of Trustees. Receipt of a Fifty Year Pin does not in itself change the member's classification of membership.

Section 8.00 Administration and Annual Program of Activities. The Board of Trustees shall adopt administrative rules of procedure necessary to carry out the duties herein described, and shall develop an Annual Program of Activities based on recommendations submitted by the Council on Planning and Development. The program of priority projects shall be considered and developed at the annual meeting of the Board of Trustees for implementation during the succeeding organizational year.

Little evidence to support withholding ERT

Estrogen replacement therapy for breast cancer survivors may be worth the risk

There is "surprisingly little" clinical information to substantiate concern that prescribing estrogen replacement therapy (ERT) for women with a history of breast cancer might activate dormant tumor cells, according to a special communication in the August 17 *Journal of the American Medical Association*. Melody A. Cobleigh MD, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Ill., and colleagues write, "We believe it is time for a change, and we believe that the time is right to study the effects of ERT in breast cancer survivors."

Estrogen replacement therapy has been shown to lessen the annoying and sometimes debilitating symptoms of menopause, and has also been shown to reduce coronary heart disease, as well as reduce fractures by maintaining bone mineral density. However, the standard of care in the United States is to discourage prescription of ERT for breast cancer survivors because of concern that ERT might activate dormant tumors and/or increase breast density and potentially delay diagnosis of breast cancer.

The researchers, who conducted a literature review of MEDLINE and CANCERLINE through the National Library of Medicine, write: "Unfortunately, the literature on the subject is limited. Most studies were retrospective. The duration and type of ERT varied, and baseline studies were often unavailable....

"The association between ERT and risk of breast cancer remains controversial, although 24 studies and three meta-analyses have been published since 1980. Collectively, the studies do not consistently demonstrate an increased risk of breast cancer among women who have ever used ERT. Studies that controlled for screening showed no difference in relative risk between cases and controls. This suggests that there may be a detection bias operating in some studies that have reported an increased risk of breast cancer among ERT users."

The annual incidence of new primary breast cancers among breast cancer survivors is 14 per 1,000 women, compared to an incidence of two per 1,000 women in the general population.

The study says: "The prohibition of ERT may diminish quality of life in breast cancer survivors. Furthermore, it is possible that the prohibition of ERT could reduce overall survival in such women by leaving them at increased risk of coronary heart disease and osteoporotic fracture....

"Women rely on their oncologists for advice on this issue, and oncologists must be broad-minded and consider all-cause mortality, not just mortality from breast cancer, when designing clinical trials. There are no published controlled clinical trials of the effect of ERT on breast cancer survival....

"Clinical trials of ERT in breast cancer survivors have been hindered in part by the maxim *primum non nocere* ('first do no harm'). Such moralizing prevents moral reflection.... In light of the lack of a detrimental effect of ERT on the health of women, we suggest a new maxim, *primum certior fi, tunc mone* ('first understand, then advise'). This review supports the ethics of a clinical trial of ERT in breast cancer survivors."

First Lady *(continued)*

liances proposed by the President and the cost of health care reform, particularly for small businesses. The Gephardt and Mitchell bills will not require businesses to purchase insurance through alliances, as originally proposed by the President. Instead, the bills will simply give small businesses and individuals the opportunity to bargain for and purchase health insurance on the same basis as large employers by allowing them to join voluntary, non-governmental purchasing cooperatives. No longer will small businesses pay as much as 35 percent more than big businesses for the same health insurance. And no longer will small businesses pay administrative costs that amount to as much as 40 percent of their health care costs, compared to five percent for big businesses.

Thank you for your support. Members in both the House and the Senate have made great progress in proposing a less bureaucratic and less governmental approach to health care reform, while preserving the President's fundamental goal of guaranteeing private health insurance to all Americans. I hope that you, as a physician and parent, will continue to work with the President to make health security a reality for all American families.

(Signed)
Hillary Rodham Clinton

Capsules

■ **St. Anthony Hospital** in Oklahoma City is finalizing plans for its annual continuing medical education seminar "Family Medicine Update 1994" (formerly "Focus on Families") to be held Friday and Saturday, November 18 and 19, 1994, at the Clarion Hotel and Conference Center in Oklahoma City. In addition to members of the St. Anthony medical staff, speakers will include John R.T. Reeves, MD, University of Vermont; Marc Cohen, MD, Mayo Clinic of Jacksonville, Fla.; and Paul S. Frame, MD, Tri-County Family Medicine, Cohockton, N.Y. In conjunction with the seminar, there will be a special dinner at the Oklahoma City Golf and Country Club and an afternoon at Remington Park. For information and registration materials, call (405) 272-6354.

■ **A caption** on page 376 of the August JOURNAL mistakenly identifies Michael J. Haugh, MD, chairman of the Oklahoma delegation to the AMA, as Dr. Edward N. Brandt. Dr. Brandt did not attend the meeting. Our apologies to both doctors for the confusion.

Reference outlines ethical obligations to patients

AMA releases 1994 *Code of Medical Ethics, Current Opinions...*

The American Medical Association has released the 1994 *Code of Medical Ethics, Current Opinions with Annotations.* The AMA code of ethics is the preeminent code of ethical conduct for U.S. physicians. It is regularly updated and overseen by the AMA's Council on Ethical and Judicial Affairs (CEJA).

The AMA ethics code outlines physicians' ethical obligations to their patients; it is also used by courts legislatures, licensing boards, and other professional societies in making, laws, deciding cases, and setting professional standards.

Current Opinions includes the AMA's seven principles of medical ethics. CEJA's interpretations of how these principles apply to the many ethical issues in modern medical practice. New and updated opinions cover a range of issues affecting the physician-patient relationship and reflect the array of technological and societal changes in health care. Some examples are:

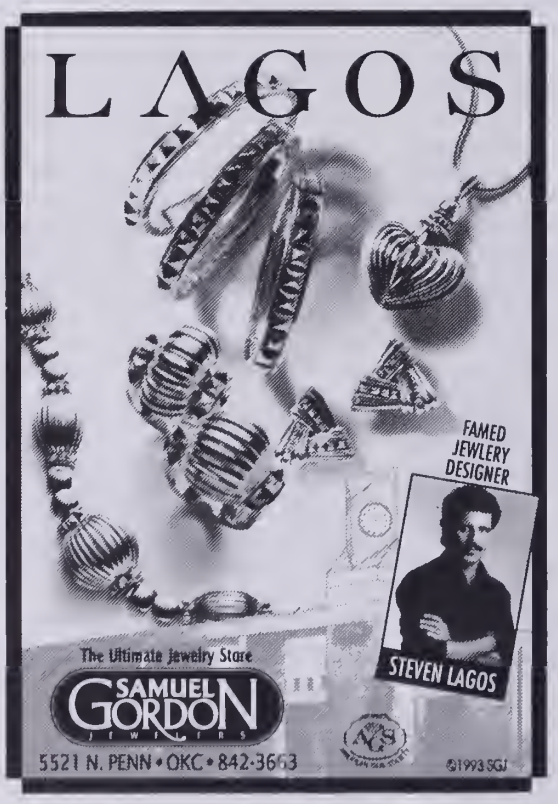
(Op 2.02) Abuse of Children, Elderly Persons, and Others at Risk sets guidelines for detecting and treating family violence. Physicians should routinely ask patients if they have been abused. Physicians also should consider battering when patients present with a number of medical complaints, particularly when physicians are treating female patients;

(Op. 2.145) Pre-Embryo Splitting (embryo cloning) procedures should be made available as an important adjunct to infertility treatment;

(Op. 2.162) Anencephalic Infants as Organ Donors delineates under what conditions it is ethically acceptable to procure the organs of anencephalic infants for life-saving transplantation;

(Op. 2.211) Physician-Assisted Suicide reaffirms the profession's opposition to physician participation in a patient's suicide. This opinion emphasizes that physicians must aggressively respond to the needs of patients at the end of life with comfort care, pain control, respect for autonomy, and good communication;

CEJA comprises eight physicians (one resident) and one medical student. CEJA Opinions are supported by reports on each topic. To order *Current Opinions,* call AMA Customer Service at 1-800-1621-8335 and request order number OP632394CH. The cost is $15 for AMA members and $35 for nonmembers.



Statement of Ownership, Management, and Circulation
(Required by 39 U.S.C. 3685)

1. Publication Title: Journal
2. Publication No: 0030-1876
3. Filing Date: Sept. 15, 1994
4. Issue Frequency: Monthly
5. No. of Issues Published Annually: 12
6. Annual Subscription Price: $20 Yr. members, $30 Yr. non-members
7. Complete Mailing Address of Known Office of Publication: 601 NW Expressway, Oklahoma City, Oklahoma County, OK 73118-6032
8. Complete Mailing Address of Headquarters or General Business Office of Publisher: 601 NW Expressway, Oklahoma City, OK 73118-6032
9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor
   Publisher: Oklahoma State Medical Association, 601 NW Expressway, Oklahoma City, OK 73118-6032
   Editor: Ray V. McIntyre, MD, OSMA, 601 NW Expressway, Oklahoma City, OK 73118-6032
   Managing Editor: Susan F. Records, OSMA, 601 NW Expressway, Oklahoma City, OK 73118-6032
10. Owner
    Full Name: Oklahoma State Medical Association
    Full Address: 601 NW Expressway, Oklahoma City, OK 73118-6032
11. Known Bondholders, Mortgagees, and Other Security Holders Owning or Holding 1 Percent or More of Total Amount of Bonds, Mortgages, or Other Securities. If none, check here: None
12. For completion by nonprofit organizations authorized to mail at special rates. The purpose, function, and nonprofit status of this organization and the exempt status for federal income tax purposes *(Check one)*: Has Not changed During Preceding 12 Months
13. Publication Name: Journal
14. Issue Date for Circulation Data Below: September 1994
15.

Extent and Nature of Circulation	Average No. Copies Each Issue During Preceding 12 Months	Actual No. Copies of Single issue Published Nearest to Filing Date
a. Total No. Copies	4752	4800
b. Paid and/or Requested Circulation		
(1) Sales Through Dealers and Carriers, Street Vendors, and Counter Sales	0	0
(2) Paid or Requested Mail Subscriptions	4074	4084
c. Total Paid and/or Requested Circulation	4074	4074
d. Free Distribution by Mail	522	525
e. Free Distribution Outside the Mail	0	0
f. Total Free Distribution	522	525
g. Total Distribution	4596	4609
h. Copies Not Distributed		
(1) Office Use, Leftovers, Spoiled	156	191
(2) Return from News Agents	0	0
i. Total	4752	4800
Percent Paid and/or Requested Circulation	88.6	88.6

16. This Statement of Ownership will be printed in the October 1994 issue of this publication
17. Signature and Title of Editor, Publisher, Business Manager, or Owner — Date

[illegible] Managing Editor — [illegible] 15, 199[illegible]

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PS Form 3526, October 1994

In Memoriam

1993

William Alfred Waters, MD ..... March 19
William Niebuhr Weaver, MD ..... March 20
Willie George Long, MD ..... March 22
William Finis Ewing, Jr., MD ..... March 31
John Robert McInnis, MD ..... March 31
Chester Randall Seba, MD ..... April 4
Edward Cotter Murray, MD ..... April 9
Eric McLain White, MD ..... April 23
John Franklin DeJarnette, Jr., MD ..... May 1
Harold Alwin Smith, MD ..... May 4
Kenneth Warren Bonham, MD ..... May 28
John Raymond Stacy, MD ..... July 6
Carl Davis Osborn, MD ..... July 11
Mark Royal Johnson, MD ..... July 12
W. Turner Bynum, MD ..... July 13
James Dougherty Martin, MD ..... July 31
Edward David Greenberger, MD ..... August 6
Horace Harold Porter, MD ..... August 15
Clifford A. Traverse, MD ..... August 20
J. Wendall Mercer, MD ..... September 2
Emma Jean Anthis, MD ..... September 27
Russell D. Harris, MD ..... September 28
Walter Lee Honska, Jr., MD ..... October 9
Ethel Maurice Walker Heras, MD ..... October 25
James Burton Pitts, Jr., MD ..... October 29
John Daniel Capehart, MD ..... November 20
Kevin James Cammack, MD ..... December 4
William Best Thompson, MD ..... December 10
Henry Grady Ryan II, MD ..... December 17

1994

Fannie Lou Leney Hayward, MD ..... January 2
Kirk Thornton Mosley, MD ..... January 3
Richard Charles Wade, MD ..... January 6
Austin Walsh Webb Haddox, MD ..... January 13
Earl Mathiews Woodson, MD ..... February 20
Tom Lamar Johnson, MD ..... March 5
Orville Main Rippy, MD ..... March 11
Minor Elliott Gordon, MD ..... March 14
George Loren Norris, MD ..... March 27
Max A. Glaze, MD ..... April 29
Winfred Aaron Showman, MD ..... May 14
Mark Daniel Holcomb, MD ..... June 1
Carter William Mathews, MD ..... June 3
Harold Ray Sanders, MD ..... June 15
Robert Bruce Howard, MD ..... June 16
Richard Warren Loy, MD ..... July 7
John Hobson Veazey, MD ..... July 11
Wesley A. Whittlesey, MD ..... July 12
Laurence Sevier McAlister, MD ..... July 19
Jon Meyer Chenette, MD ..... August 4
Jess Duval Herrmann, MD ..... August 16

Deaths

Jon Meyer Chenette, MD
1944 - 1994

OSMA Life Member Jon M. Chenette, MD, died August 4, 1994. Dr. Chenette was born in Beloit, Wisc., and earned his medical degree at the University of Tennessee, Memphis, in 1967. After completing an internship at Cook County Hospital in Chicago, and spending a year in the U.S. Navy, he began an orthopedic surgery residency at Northwestern University, completing it in 1975. His Oklahoma license was issued the following year when he established a practice in orthopedic and hand surgery in Okmulgee. In 1982 Dr. Chenette was named chief of staff at Okmulgee Memorial Hospital. Injuries in a 1984 plane crash forced his early retirement.

Classifieds

Classified ads are 50 cents a word, with a minimum of $25 per ad. A word is one or more characters bounded by spaces. Box numbers will be assigned upon request and will add 6 words to the total. Payment must accompany all submissions. Orders will NOT be accepted via telephone or fax. Mail ad with payment to OSMA JOURNAL, 601 Northwest Expressway, Oklahoma City, OK 73118. Deadline is the first of the month preceding the month of publication.

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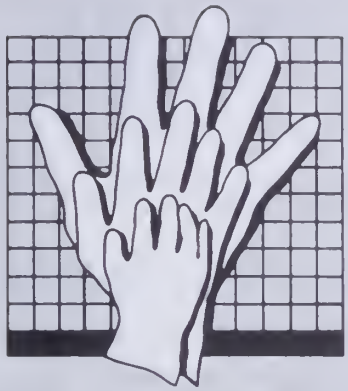

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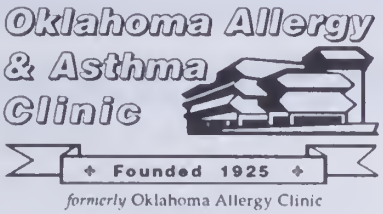

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
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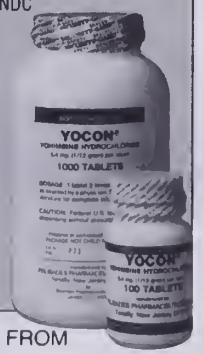

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Staying Away from the Edge

I've heard a story of a little boy who fell out of bed. When his mother asked him what happened he answered, "I don't know. I guess I stayed too close to where I got in."

It is easy to do the same thing with membership in an organization. What happens when we stay where we got in and never move? We miss all the benefits of belonging. By taking advantage of all the benefits of belonging to the Oklahoma State Medical Association Alliance (OSMAA), the boring can become bold, the doubtful can become daring, and the apathetic can become assertive.

The Oklahoma State Medical Association Alliance is a collection of many different people with many different talents brought together by a common goal of promoting good health. Alliance members are given a myriad of opportunities for growing. Three areas especially important to personal growth in the Nineties are communication, companionship, and compassion.

Communication is the key to an effective legislative voice. The year 1994 will be long remembered as the year for the term *health care reform*. Physicians' spouses should be highly involved in all areas of legislation that will affect the well-being of their spouse's practice. Interaction with congressmen by way of letter, phone call, or a day at the capitol are all tools by which OSMA Alliance members communicate their views. As alliance members become aware of legislation affecting the medical community, the indifferent become involved!

Another objective of the alliance is to be a support group for medical families. Physicians and their spouses face a different lifestyle than is common to most marriages. By joining the alliance you can interact with other people who share your special concerns. Alliance activities give its members the opportunity to combine individual talents into worthwhile projects, and soon acquaintances become allies!

Compassion is an attitude which all physicians should possess. We, the alliance, believe this attitude should encompass our activities as well. The health and quality of life for our communities are improved daily by alliance projects. The problem of family violence affects over 60 million families in America today. Few organizations are as concerned about this and other health-related issues as the OSMA Alliance. Even if you cannot be actively involved, your dues will mean those who can will continue to make a difference on the issues that concern all of us.

Membership means that the complacent become compassionate. We invite you to bring your talents and influence to our organization. Don't rest on the edge like the little boy who fell from the bed. Become involved. We have a lot of growing to do. Membership is open to the spouse of any physician who is a member of the OSMA. In counties where there is no organized county alliance, a spouse may join as a member-at-large. For membership information, call Judy Lake at the OSMA offices, (405) 843-9571.

—Abby King
OSMAA First Vice-President

■ **1-800-833-6354 is the toll-free hotline set up by the AMA** POWER Network to provide physicians with daily updates on the status of health system reform in Congress. The POWER (Physicians Organized to Work for Effective Reform) Network hotline will supply the latest information on House and Senate floor debate as well as clear instructions on how the caller can have maximum effect on important upcoming votes.

■ **Oklahoma City internist Jerry Vannatta, MD, has been** appointed by Governor David Walters to a newly created position on the Oklahoma Health Care Authority. Dr. Vannatta's four-year term will expire in 1998. The position was created by SB 650 in the last legislative session. Dr. Vannatta is a professor of medicine at the OU College of Medicine and director of medical education at Presbyterian Hospital. Also serving on the authority are Theodore J. Brickner, Jr., MD, Tulsa radiologist, and Garth L. Splinter, MD, a family practitioner in Oklahoma City. Dr. Splinter is the authority's CEO.

■ **One-third of AMA dues for 1995 will not be tax deduct**ible because of association lobbying expenditures. This amends an earlier announcement that 23% of 1994 dues could not be deducted as a business expense. "No portion of any dues paid to AMPAC or a state medical society PAC may be deducted as a business expense for federal income tax purposes," the AMA said. "Medical society and PAC dues also cannot be deducted as a charitable contribution for federal income tax purposes."

■ **"Oklahoma Conference on Fetal and Infant Mortali**ty: A Community Approach to Mortality Prevention" is the title of a two-day meeting to be held Thursday and Friday, November 3 and 4, 1994, at the MetroTech Conference Center in Oklahoma City. The event will feature plenary speakers Henry A. Thiede, MD, of the American College of Obstetricians and Gynecologists and chair of the Department of Gynecology at the University of Rochester; Timothy D. Dye, PhD, director of the Perinatal Data System at the State University of New York at Syracuse; and Ronald A. Chez, MD, professor of obstetrics and gynecology at the University of South Florida.

Concurrent sessions will include Implementing Fetal and Infant Mortality Review in Your Community; Demonstration of a Community Review Panel; Grief and the Family; role of the Physician in Fetal and Infant Mortality Review; Family Interviewing; Abstracting Medical Records; Importance of Complete Medical Records and Birth Certificates; Impact of Unintended Pregnancy and Family Planning; and Advocacy for Decreasing Fetal and Infant Mortality.

The event is sponsored by the Oklahoma Fetal and Infant Mortality Analysis Program, Department of Pediatrics, University of Oklahoma Health Sciences Center (OUHSC); Oklahoma State Department of Health/Maternal and Infant Health and the Planning and Evaluation Division; and the OUHSC College of Public Health.

For registration information, contact the OU College of Public Health, (405) 271-2342.

■ **The American Medical Association Hospital Medical** Staff Section (AMA-HMSS) will hold its 24th Assembly Meeting December 1 through 5, 1994, at the Sheraton Waikiki Hotel in Honolulu. Aside from participating in the development of AMA policy, medical staff representatives will have an opportunity to network with colleagues, talk with the AMA Board of Trustees, and hear the latest news and information on health system reform.

The AMA-HMSS has been asked to participate in the AMA's Federation Consortium Study, which is intended to uncover useful information for developing ways to increase membership, member participation, and advocacy as well as improve communications, medical society performance, and resource utilization. The AMA-HMSS has been asked to participate because it effectively represents grassroots physician concerns. Input from each HMSS representative also will be extremely valuable in defining organized medicine in the future. The 1994 Interim AMA-HMSS Assembly Meeting Education Program will host the consortium study on Friday, December 3, from 2:30 to 5:30 PM in Honolulu.

■ **The OSMA Hawaii trip in conjunction with the AMA** Interim Meeting is set for December 4 through 11. The first 3 days will be in Honolulu at the AMA host hotel—the Hawaiian Hilton Village. The last 4 days will be at the Mana Lani Bay Hotel on the island of Hawaii. There are special rates on airfare and hotel packages through OSMA Member Services and Seagrave Travel. Contact Seagrave for information and booking, 1-800-522-9212 or in Oklahoma City (405) 841-4464.

■ **The National Rural Health Association recently pub**lished a comprehensive rural health resources directory. The directory is designed to facilitate contact among organizations, agencies, and individuals working to improve the delivery of rural health care services. It lists such things as federal, national, and state organizations; offices of rural health hospital associations; rural health research centers; and area health education centers. Listings are organized by state within each category and are indexed by state and type of organization. The *Rural Health Resources Directory—1994* costs $10 per copy. To order, send payment to Publications Department, NRHA National Service Center, One West Armour Blvd., Suite 301, Kansas City, MO, 64111.

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POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscriptions to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single copies are $3 per copy, prepaid, subject to availability.

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Paul Patton, executive director of the Tulsa County Medical Society, provided this last look at autumn in Oklahoma.

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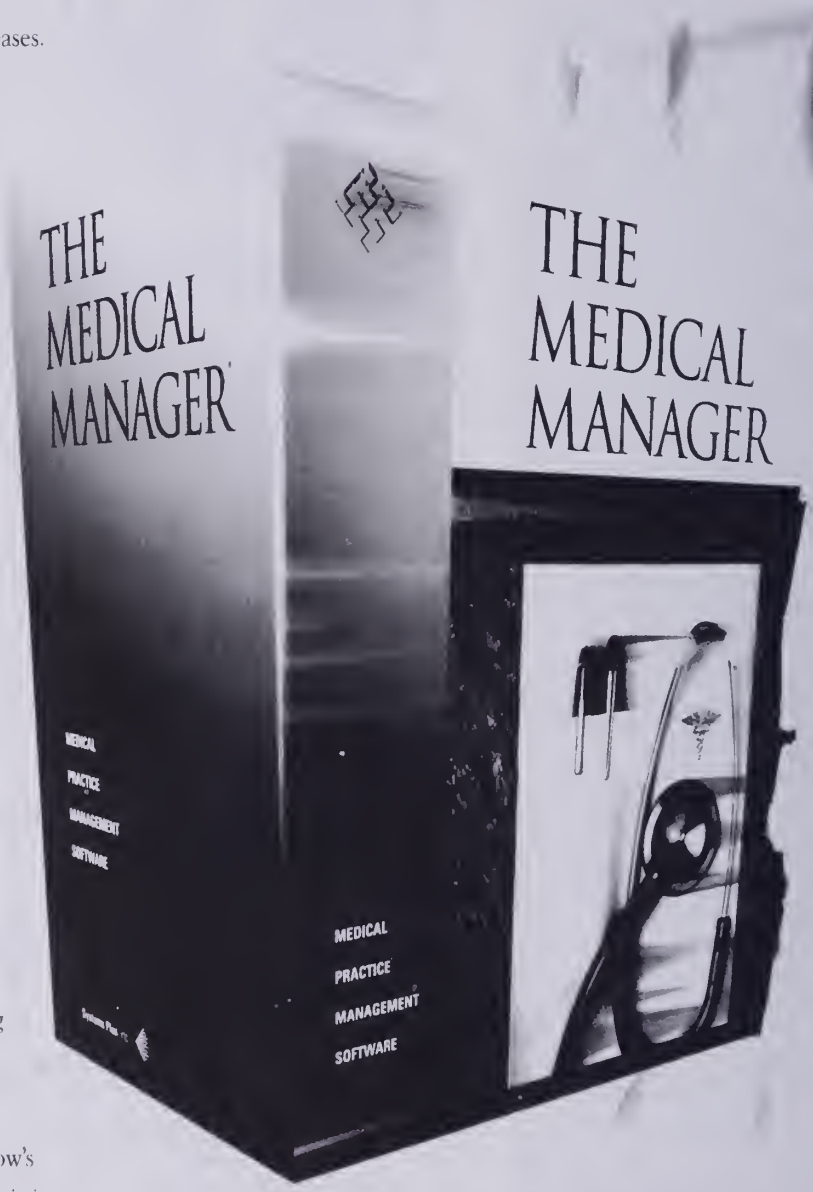

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Citizens or Sinners?

We all have, or have had, grandfathers and grandmothers. Most of us have, or will have, grandchildren. Thus we are the existent link between what has been and what will be in the future. In addition to our role as protoplasmic relay messengers in a biological system, most of us are the instrumentality that transmits the cultural values of our forebears to our progeny.

We have not been vested with control over the nature of the DNA that we are privileged to transmit. But we can use our experiences and our judgment in the selection of the cultural values that we transmit to our progeny. By the act of committing living time and energy to our enterprises, we demonstrate what has value to us, and the projects then become the nucleus of a value system for our children and grandchildren.

Among those cultural values we citizens of the United States have received from our forefathers (and foremothers) is the amazing grace of a constitutional republic, with more political control over our own lives than any tribe in the world. From the actions of our forebears we also learn that every generation of citizens has the responsibility to protect, preserve, and renew both the freedom and the republic, and to teach the children those values that perpetuate citizen liberty.

Regular democratic elections are essential to the preservation of freedom's creed, and we take note that biennially our republic's election cycle recurs and that it purges and renovates. Some public servants are cast out of office and others are returned to their offices. Our society's public policy debates are recast and re-focused by the periodic election, and new ideas and initiatives come to be advanced.

The election results sadden some of us and gladden some of us. But whether we as individuals are Demo-publicans or Republicrats, we each remain indebted to our forebears and progeny to keep the republic working. And that means that each of us should take the initiative to establish a direct connection to those functionaries who are now empowered to operate the government.

A basic first step is to cast a carefully considered vote for those candidates with a philosophy that is personally compatible. Then the successful candidates need to hear the views and opinions of those whom they represent (even those who did not vote for them). Every year there are several hundred bills introduced in the Oklahoma Legislature that affect medical practice and health care delivery. Every year bad bills become law because the legislators heard zilch from their physician constituents.

If we are to be good citizens we will make contact with our legislators and offer to be informative and helpful in the consideration of bills with medical significance. We will also make ourselves available to our OSMA lobbyists and staff to help them in their responses to particular legislative problems.

Nearby, on page 511, is reprinted a "prescription" written by Representative Don McCorkell that sagely discusses the matter of physician (citizen) interaction with the legislative process. Each of us who is to be a good citizen will follow the "prescription" with care.

Ray V. McIntyre, M.D.

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The MCO/IPA that so many of us have been working on for the past year is about to become a reality. Now it is time for you to act. Soon you will be receiving a packet in the mail which will fully describe the program. All of this is the product of countless meetings of the OSMA, the Board of Trustees, and two sessions of the House of Delegates. We also traveled across the state talking to physician groups, county medical societies, and some speciality societies trying to arrive at a consensus on what should be done to help our members cope with the Health System Reform proposed by the Clintons and state legislators, and demanded by medical care purchasers. Some thought we were too late; some were concerned about our ability to compete; some were afraid we would fail. But almost all wanted the OSMA to develop a reasoned and practical response. As a result, the Board of Trustees commissioned the Garvey Group to conduct a feasibility study. The results were positive and the House of Delegates, in April, approved the plan (BOT Report B). The interim MCO/IPA boards have followed the House's instructions and the plan has been implemented to the point of capitalization.

What we have been seeking is the best solution to a national movement driven by government and business to lower medical care costs and to change the method of payment for medical services. Our primary goal has been to keep control of all medical decisions with the doctors and their patients.

A wordy mission statement has been approved by the boards and it says: The purpose of the plan is to provide quality health care to the people of Oklahoma by maintaining the traditional relationship between the patient and physician with mutual responsibility to each other, by providing the highest quality medical care at an affordable cost, by preventing further fragmentation of the health care system, by assuring that health policy and the delivery of health care stay in the hands of physicians and their patients, by establishing a cost-effective administrative system, keeping more dollars for health care, by keeping the premiums normally distributed to non-medical entrepreneurs to provide benefits for the participant, and by establishing a managed care plan that any good physician who is a member of the OSMA or OOA can join, and one in which any patient may exercise his or her choice of physician.

From all of that lengthy mission statement, please remember these four points: (1) We must preserve the traditional relationship between the patient and physician. (2) We must provide the highest quality medical care at an affordable cost. (3) We must have competitive premiums while fairly compensating the physicians rather than profiting non-medical entrepreneurs. (4) The patients must have their choice of physicians.

None of the operating managed care plans with which we are familiar meet all of these criteria. We think our MCO/IPA can. The interim boards have adopted the following methodology:

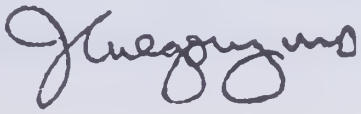
First, the formation of an Independent Practice Association. Its name shall be Oklahoma Physicians Network (OPN). Membership will be available to all members of the OSMA and the OOA, approximately 5,000 physicians. OPN is a non-profit corporation. It has no stock and is currently governed by the interim board. The bylaws adopted by the interim board describe in detail the election and composition of the new board, which represents fairly all segments of our profession and gives the OOA fair representation. OPN will enter into contracts with participating physicians who meet the eligibility and credentialing requirements, and who agree to purchase stock in the MCO. OPN will contract with the MCO to provide the full range of physician services for its enrollees.

Second: The creation of a for-profit Managed Care Organization whose name shall be PROklahoma Care will be accomplished through the sale of stock to members of OSMA and OOA who are residents of Oklahoma. Each member may purchase one share of voting stock and all stockholders will have equal voting privileges. In other words, *one share, one vote.* The corporate papers have been filed and the interim board has approved bylaws that set out in detail the election of and representation on the new board.

Initially, PROklahoma Care will apply for a license from the state health department as an HMO. It is envisioned that in time, and as the market demands, a full line of managed care products will be made available to Oklahoma patients, employers, and organizations. Like an insurance company, PROklahoma Care will take risks and have administrative expenses and thus will need capital, estimated to be around 6 million dollars. PROklahoma Care will be an independent corporation owned by its stockholders and will have no formal corporate relationship with the OSMA. Because of the wishes of its members, the association has facilitated the formation of both corporations, advanced funding, and furnished staff support, as well as paid for consultants. OSMA will have no control of or financial obligation for PROklahoma Care's activities. From the monies raised by the sale of stock, OSMA will be repaid the dollars it has made available so far. At first, neither corporation will have full-time staff. All marketing and administrative services will be contracted through C.L. Frates and Company, Inc. They have agreed to a fee of 10% of gross premium for providing a turnkey management service, almost identical to their contract with PLICO. This will hold overhead to a minimum and will be an important factor in our ability to negotiate contracts with prospective purchasers.

Let me digress for a moment and bring you up to date on HMOs in Oklahoma. There are at least 6 HMOs licensed and operating in our state. The largest is Pacificare and its subsidiary, MultiMed, with approximately 118,600 enrollees, or 4% of the market. PruCare has approximately 2%, BlueLincs 1%, and Cigna and Community Care less than 1%. Collectively they have around 225,000 enrollees, or roughly 8% of the market, barely larger than the largest insured group in the state, the State Employees Group, with about 212,000 members. Most health care experts project that within the next five years, 40% to 60% of Oklahoma's insured populace will be enrolled in managed care plans. That is approximately 1.6 million patients, or 8 times the number enrolled now. These numbers as well as the opinions of outside experts support our assumption that there is and will be a demand in the months ahead.

So let me repeat myself, THE TIME IS NOW. We have a window of opportunity, a golden opportunity to preserve the most sacred of relationships, the Patient-Physician Relationship. By forming the IPA/MCO we can control our own destiny. These entities will be totally, 100% physician owned, directed, and controlled. You and I will elect the boards that will make the decisions regarding health care delivery in these entities. For this venture to be successful we all must capitalize these companies, and we must be willing to subject ourselves to very close review of both the quality and the quantity of the health care we deliver. You must not look upon your dollars as a get-rich-quick scheme. Rather, you must view your dollars as an investment in your future and the future of your profession. You, the members of the OSMA, have asked us to do this. Now is the time for each of you to do your part to preserve our future, to provide an arena in which we can practice high quality, cost effective medicine while preserving the Patient-Physician Relationship. Our more senior members formed PLICO, and it has and will continue to serve us well, just as OPN and PROklahoma will in the future. THE TIME IS NOW. Good day my friends...



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AIR FORCE

Acute Cerebral Edema as Part of the Syndrome of Hepatic Encephalopathy in an Individual with Chronic Liver Disease: A Case Report

N. Jabbour, MD; D. Karavias, MD; D.H. Van Thiel, MD

An unusual case of acute cerebral edema as part of the syndrome of portal systemic encephalopathy in an individual with established chronic liver disease is reported. Several episodes of edema occurred with clinically important increases in the blood ammonia level.

From the Pittsburgh Transplant Institute, Department of Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pa., and the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Okla.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, OK 73112.

Increased intracranial pressure is present in more than 80% of patients with fulminant hepatic failure, but its presence is not well documented in patients with chronic liver disease. Herein we report a patient with cirrhosis who developed severe hepatic encephalopathy after an upper gastrointestinal bleed. This encephalopathy was complicated by seizures and progressive cerebral edema documented both by computed tomographic (CT) scanning of the head and direct measurement of the cerebrospinal fluid pressure.

Case Report

D.R. was a thirty-year-old man with end-stage cirrhosis secondary to hepatitis B who had recently experienced two episodes of upper gastrointestinal bleeding treated with variceal sclerotherapy. He was admitted to the hospital for possible liver transplantation after a third episode of upper gastrointestinal bleeding. His history was significant for increased ascites and fatigue. On admission his physical examination documented normal vital signs and a completely normal neurological exam. He had ascites; a large, easily palpable spleen; and mild pedal edema.

His laboratory studies demonstrated a total bilirubin of 2.5 mg/dl, GGTP of 169 U/L, SGOT of 83 U/L, SGPT of 91 U/L, albumin of 2.4 G/dl, PT of 14.1 seconds, platelet count of 47,000 per mm^3, hematocrit of 36%, BUN of 20 mg/dl, creatinine of 0.7 mg/dl, and an ammonia level of 263 ug/dl (normal <50).

An upper endoscopy documented actively

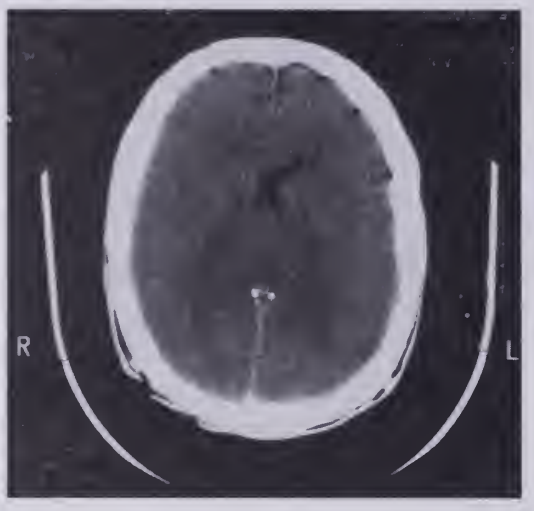

Figure 1. CT scan of the head demonstrating marked cerebral edema with loss of cortical sulci and loss of third and lateral ventricular volumes.

Figure 2. CT scan of the head obtained earlier in the same patient showing well-preserved cortical sulci and normal lateral ventricular appearance.

bleeding esophageal varices which were sclerosed with two injections of 1.5 cc of ethanolamine with control of the bleeding. The patient was transferred to the intensive care unit and was treated with oral lactulose and a continuous infusion of octreotide acetate. His ammonia level decreased to 68 ug/dl within 24 hours.

Two days later the patient had a recurrent episode of gastrointestinal bleeding which led to a brisk deterioration of his mental status. He was intubated and given 2 units of blood. A repeat upper endoscopy revealed recurrent variceal bleeding which was treated with sclerotherapy. Repeat liver chemistry studies were similar to the values observed initially except for the bilirubin and ammonia levels, which had increased to 6.9 mg/dl and 236 ug/dl respectively.

Three days later the patient's ammonia level had decreased to a value of 74 ug/dl; he was extubated and his ascites was drained percutaneously. His mental status was normal. He was listed for orthotopic liver transplantation and transferred to the floor with a normal mental status, stable hemodynamics, and liver chemistry, which were similar to those done on admission.

Two days later the patient had a fourth episode of upper gastrointestinal bleeding and was transferred back to the intensive care unit. At this time his mental status was normal and his vital signs were stable. The patient was electively intubated using midazolam and fentanyl prior to an upper endoscopy. His ammonia level was 293 ug/dl and his total bilirubin was 6.6 mg/dl. He was transfused with 4 units of red blood cells. Forty-eight hours after his last episode of upper gastrointestinal bleeding, while he was still intubated because of a depressed mental status, he experienced tonic-clonic seizures localized to his tongue, left arm, left jaw, and left side of his neck lasting for 3 minutes. The seizures were controlled with 10 mg diazepam IV and 1 g of IV diphenylhydantoin. Post ictal neurological exam revealed no response to any stimulus. His brain stem reflexes were absent (no gag reflex, negative doll eyes, and negative cold caloric stimulation). His pupils were 2 mm in diameter and not reactive to light. His motor tone was flaccid.

A CT scan of the head (Fig. 1) revealed generalized swelling without any mass effect; this was in contrast to his admission CT scan of the head which was unremarkable (Fig. 2). An electroencephalogram was obtained and revealed marked suppression of all cerebral activity. The patient's oxygen saturation was always 100% on 40% FI02, his ammonia level was reduced to 45 ug/dl. No clear cause of his seizures could be identified. A lumbar puncture documented an increased intracranial pressure (opening pressure of 46 cm of water). The cerebral spinal fluid glucose concentration was 156 mg/dl, protein was 61 mg/dl, and contained 5 WBC per mm^3.

The patient was started on furosemide and mannitol infusions. Because of his coagulopathy, invasive intracerebral pressure monitoring was avoided. A repeat electroencephalogram two days later revealed mild improvement. Although the patient recovered brain stem reflexes, he continued to have a persistent decorticate posture. A repeat CT scan of the head failed to document any improvement.

The patient underwent a TIPS (trans intrahepatic portal systemic shunt) procedure to control his recurrent bleeding. This resulted in reduction of the wedged portal venous pressure gradient from 25 cm of water to 12 cm. Despite optimal medical control, his neurological function never returned to normal and his hospital course was complicated by generalized sepsis and spontaneous bacterial peritonitis. The patient died one month after admission. An autopsy was refused by the family.

Discussion

Although cerebral edema is a well-known complication of fulminant hepatic failure[1] and one of the most important prognostic factors in patients with fulminant hepatic failure, it is not well documented in chronic liver disease. Two similar cases were reported recently with a documented increase of intracranial pressure associated with chronic liver failure.[2] The severity of chronic hepatic encephalopathy correlates well with the blood ammonia level. Cerebral edema in chronic liver disease is unusual if not rare.

In the case reported, a blood high ammonia level (up to 293 ug/dl) at three different times was the only metabolic abnormality found. Although he had recurrent episodes of gastrointestinal bleeding requiring multiple transfusions, he was at no time hypoxic or hemodynamically unstable. Therefore, anoxic injury of the brain was not present. The normal protein and cell counts in the cerebrospinal fluid make an infectious cause of the cerebral edema unlikely. Fentanyl and midazolam are not known to produce any increase in intracranial pressure. It is interesting to note that the patient's liver chemistry was inconsistent with an acute hepatic injury during his hospital course and the mild increase in his bilirubin level was most likely related to his blood transfusions. Hyperammonia was the only finding associated with cerebral edema in this patient. Thus, the recurrent episodes of severe hyperammonia are most likely the cause of his cerebral edema. In animal studies, hyperammonemia can be shown to produce cerebral edema.[3,4] The recurrent gastrointestinal bleeding experienced by this patient probably produced intermittent increases in his ammonia level.

The severity of chronic hepatic encephalopathy correlates well with the blood ammonia level.

Conclusion

This report documents that hepatic encephalopathy in chronic liver disease can be complicated by cerebral edema leading to brain stem injury.

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The Authors

Nicholas Jabbour, MD, is assistant professor of surgery at the University of Pittsburgh (Pa.) School of Medicine.

Dionysieas Karavias, MD, is a transplant surgeon at the Pittsburgh Transplant Institute, University of Pittsburgh School of Medicine.

David H. Van Thiel, MD, is the medical director of transplantation at the Oklahoma Transplantation Institution, Baptist Medical Center of Oklahoma, Oklahoma City.

Microbiological Flora and Nail Polish: A Brief Report

Ghazi M. Rayan, MD; D.J. Flournoy, PhD; Pam Schlageter, RN

Removing nail polish and prosthetic nails from operating room (OR) personnel prior to scrubbing and from patients prior to hand surgery is recommended but not practiced in many hospitals. There is concern that nail polish can act as a vehicle for the transfer of infectious agents.

This study was designed to determine the incidence of microbiological flora of nail polish in a clinical setting.

From Baptist Medical Center and University of Oklahoma Hospitals, Oklahoma City, Oklahoma.

Direct correspondence to Ghazi M. Rayan, MD, Physicians Building D, 3366 Northwest Expressway, #700, Oklahoma City, OK 73112.

Materials and Methods

Twenty-one hands of 19 women were the subject of this study. There were 9 patients and 10 OR personnel including nurses and technicians. The patients were undergoing a variety of hand surgery procedures, but none were undergoing surgery for hand infection. None of the OR personnel had a recent history of hand or perionychial infection. The OR personnel were asked not to remove their nail polish prior to scrubbing their hands. The patients' nail polish was not removed prior to prepping the hand.

Using sterile technique, cultures were obtained from the OR personnel during the first cases of the day. Each underwent the usual 10-minute scrub. Each patient underwent the usual povidone-iodine soap and solution preparation of the operative hand for ten minutes. Following scrubbing and preparation, samples of nail polish were scraped with a #10 blade from all fingers of patients and OR personnel. The remaining polish was cleaned with polish remover. Povidone-iodine solution was used again on OR personnel and patients' hands, and the surgery proceeded as planned.

A total of 21 specimens were submitted to the microbiology laboratory for cultures. Fingernail polish scrapings were placed into 5.0 ml of trypticase soy or thioglycollate broth in a screw-cap tube. The inoculated broth was incubated at 35°C for 5 days. Isolates were identified by standard microbiological procedures and antimicrobial susceptibility testing was performed using disc agar diffusion.

Results

Of the 21 specimens, 5 had bacterial isolates, 2 of these were *Staphylococcus epidermidis*. The remaining 15 specimens had no growth. Results of antimicrobial susceptibility tests are shown in Table 1.

Table 1. Culture Results from Fingernail Polish Shavings

Hand	Individual	Culture Results
1	1	No growth
2	2	*Bacillus* sp.
3	3	No growth
4	4	No growth
5	5	No growth
6	6	No growth
7	7	*Acinetobacter calcoaceticus* var. *Iwoffi*[a]
8	8	No growth
9	8	*Staphylococcus epidermidis*[b]
10	9	No growth
11	9	No growth
12	10	No growth
13	11	*Staphylococcus epidermidis*[c]
14	12	No growth
15	13	No growth
16	14	No growth
17	15	No growth
18	16	No growth
19	17	No growth
20	18	No growth
21	19	*Propionibacterium* sp.

[a]susceptible to ampicillin, amikacin, gentamicin, sulfamethoxazole-trimethoprim and ciprofloxacin and resistant to cefoxitin, cefoperazone, cephalothin and intermediate to piperacillin
[b]susceptible to gentamicin, oxacillin, penicillin, cephalothin, chloramphenicol, clindamycin, tetracycline, sulfamethoxazole-trimethoprim, vancomycin and resistant to erythromycin
[c]susceptible to gentamicin, oxacillin, penicillin, cephalothin, chloramphenicol, clindamycin, erythromycin, tetracycline, sulfamethoxazole-trimethoprim and vancomycin

Discussion

Some medical centers allow nurses and OR technicians to wear nail polish and artificial nails in the OR. The Association for Operating Room Nurses (AORN) recommends that "finger nails should be free of nail polish and trimmed short."[1] One study[2] showed that nail polish was not associated with increased microbial count of the finger nails; however, the study method included a surface swab rather than scraping the nail polish.

In our study nail polish scraping cultures were obtained immediately following hand scrubbing from patients and OR personnel. In spite of preoperative hand scrubbing in the usual sterile manner, 5 of 21 specimens had bacterial growth. Although *Staphylococcus epidermidis* is often considered a harmless skin flora, it can become a pathogen and cause systemic infection.

In a previous study we were able to isolate microorganisms from fingernail trimmings of all patients who underwent preoperative hand scrubbing.[4] The majority of the patients (95%) had a moderate to heavy growth of *Staphylococcus epidermidis*. We concluded that *Staphylococcus epidermidis* is an inhabitant of the fingernails and resides in the undersurface of nails adjacent to the hyponychial skin. We also concluded that patients' fingernails should be trimmed prior to surgery. The presence of nail polish has no favorable effect on the nail flora because it covers the dorsal surface of the nail. To the contrary, it may entrap bacteria over the nail surface. Some patients have transparent nail polish, which is not recognized easily, and therefore these patients may undergo surgery without removal of the nail polish. Nail polish may crack and peel during mechanical scrubbing, which could expose an underlying contaminated nail surface during surgery.

Although the majority of nail scrapings did not have any growth, isolation of bacteria from nearly 24% of volunteers and patients is a cause of concern. Removing nail polish from patients undergoing surgery and OR personnel assisting in surgery might further decrease the chance of wound contamination and subsequent complications. It is possible that the presence of *Staphylococcus epidermidis* within nail polish scrapings may be a contaminant from the fingernails or adjacent cutaneous flora prior to polish application.

Our study does not address the nature of prosthetic nail microbiologic flora. Previous studies showed that prosthetic nails are greater carriers of Gram negative microorganisms and fungi than are natural nails.[3,5,6] Gaps between natural and artificial nails may trap moisture, which creates a milieu for bacterial growth.

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The Authors

Ghazi M. Rayan, MD, is clinical professor of orthopedic surgery at the University of Oklahoma College of Medicine. He also has a private practice in upper extremity, hand, and microsurgery in Oklahoma City.

Dale J. Flournoy, PhD, is director of clinical microbiology at the Veterans Affairs Medical Center in Oklahoma City.

Survival Following Organ Transplantation in an Oklahoma Institution, 1985-1993

D.K.C. Cooper, MD, PhD; E.N.S. Samara, MD; L. Mieles, MD; H. Merhav, MD; N. Imes, MD; D. Van Thiel, MD; N. Zuhdi, MD

Since 1985, a total of 413 patients have undergone 439 solid organ transplants at the authors' institution. The current actuarial one-year survival rate of patients undergoing heart, kidney, lung, or liver transplantation at our center is 94%, 90%, 87%, and 91%, respectively. Five-year survival of heart and kidney recipients is 80% and 75%, respectively. In view of these excellent results and the excellent quality of life that successful organ transplants provide patients with end-stage organ failure, every possible effort should be made to increase organ donation.

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Oklahoma.

Direct correspondence to D.K.C. Cooper, MD, PhD, Oklahoma Transplantation Institute, Baptist Medical Center, 3300 Northwest Expressway, Oklahoma City, OK 73112.

Heart and kidney transplantations have been performed at the authors' institution since 1985 and 1987, respectively. A single-lung transplant program was initiated in 1990 and a liver transplant program in 1992. As of December 31, 1993, a total of 413 patients have undergone 439 organ transplants here (Table 1). We present a brief review of the overall results of these four programs. Actuarial survival of patients has been determined using the Kaplan-Meier test.

Heart Transplantation

Since March 1985, one hundred seventy-six patients have undergone heart transplantation. One hundred seventy of these 176 were orthotopic transplants and 6 were heterotopic transplants (Table 1). Four of the 176 procedures were retransplants for acute or chronic graft rejection. The major indications were ischemic heart disease (57%) and idiopathic (dilated, viral) cardiomyopathy (33%). Other less common indications for heart transplantation included rheumatic heart disease, congenital heart disease, cardiac trauma, post-partum cardiomyopathy, and cardiomyopathy from Adriamycin toxicity.

No fewer than 100 (57%) of those awaiting heart transplantation were being supported by inotropic drugs and/or an intra-aortic balloon pump with or without associated positive-pressure ventilation in an intensive care unit immediately prior to their transplant procedure.[1] One patient had been on a left ventricular assist device for a total of seven weeks before transplan-

Table 1. Patients Undergoing Organ Transplantation, 1985-93				
	Heart	Kidney	Lung	Liver
Total	176	193	15	55
Primary Transplant	172	173 [cadaveric 130 (78%)] [living-related 43 (22%)]	15	54
Secondary Transplant	4	20 (20%)	0	1
Male:Female	143 (81%) : 33 (19%)	114 (59%) : 79 (41%)	8 (53%) : 7 (47%)	26 (47%) : 29 (53%)
Mean Age (Range) / Years	51 (15-68)	32 (16-66)	54 (48-63)	50 (26-66)

tation was performed. Details of the clinical management of heart transplant recipients at our center have been published previously.[2,3]

Overall patient survival following heart transplantation has been90% at one year and 80% at five years (Table 2). One patient who developed renal failure almost three years after heart transplantation subsequently underwent successful kidney transplantation and currently remains well five years post-heart transplant and two years following kidney transplantation.

Of the last 100 consecutive heart transplants, performed since July 1989, patient survival has been 94% at one year and 92% at two years.

Table 2. Actuarial Patient Survival (%) Following Organ Transplantation, 1985-93				
Survival	Heart	Kidney	Lung	Liver
30 days	97% (98%)*	100%	93%	98%
3 months	93% (96%)	98%	87%	93%
1 year	90% (94%)	90%	87%	91%
2 years	85% (92%)	87%	73%	
5 years	80%	76%		

*Figures in parentheses represent survival of the most recent 100 consecutive heart transplant patients (July 1989-December 1993).

Kidney Transplantation

One hundred ninety-three kidney transplants have been performed here since January 1987. One hundred thirty (68%) were primary transplants, while 20 (10%) were retransplants. These 150 kidney transplants were performed using cadaveric donors (Table 1). An additional 43 patients (22%) received kidneys from a living-related donor (parent 12, sibling 23, child 8).

One of these 193 patients had previously had a heart transplant (performed almost three years earlier), while a second patient underwent successful simultaneous combined liver/kidney transplantation.

The major indications for kidney transplantation were diabetic nephropathy (25%), chronic glomerulonephritis (14%), and polycystic disease (14%). Other less common indications included hypertensive nephropathy, nephrosclerosis, membranous and focal glomerulonephritis, lupus nephritis, and familial nephropathy.

Actuarial patient survival following kidney transplantation is shown in Table 2. Primary graft survival at one year and five years has been 90% and 76%, respectively.

Lung Transplantation

Fifteen patients at our institution have undergone single-lung transplantation for chronic obstructive pulmonary disease (n=14) or interstitial fibrosis (n=1) since June 1990 (Table 1). The initial hospital survival has been 93%. The overall one- and two-year survivals have been 87% and 73%, respectively (Table 2).[4]

Liver Transplantation

Fifty-four patients received a total of 55 orthotopic liver transplants between October 1992 and December 31, 1993 (Table 1). One patient required a second transplant. The main indications for liver transplantation were post-necrotic cirrhosis secondary to either a chronic viral infection (39%) or autoimmune chronic active hepatitis (26%), primary biliary cirrhosis (9%), and alcohol-associated cirrhosis (9%). One patient

The major factor limiting a wider application of this form of therapy is the number of organ donors that become available.

was transplanted for fulminant hepatic failure. The remaining 8 patients (15%) were transplanted for a variety of uncommon conditions.

Eight (15%) of the potential liver recipients were hospitalized in the intensive care unit immediately prior to liver transplantation (UNOS [United Network for Organ Sharing] status 4). Fifteen (28%) required continuous hospitalization outside an intensive care unit setting (status 3), and the remaining 31 (57%) were ill at home immediately prior to their transplant (status 2).

Four patients died during the first post-transplant year. The remaining 50 patients are all alive and well. The one-year actuarial survival for this group of orthotopic liver transplant patients is 91% (Table 2). One of these patients underwent successful simultaneous combined liver/kidney transplantation.

Discussion

The solid organ transplant programs are steadily expanding their activity, with a total of 117 patients undergoing organ transplantation in the last 12-month period between January and December 1993 (liver 52, kidney 41, heart 17, lung 7). With regard to patient and graft survival, the results of all four transplant programs compare favorably with published national and international results.

For example, figures reported from the Registry of the International Society for Heart and Lung Transplantation show a 30-day mortality following heart transplantation of 8% whereas in the reported series the comparable mortality in the last 100 consecutive patients has been only 2% (Table 2). Similarly, the mean one-year and five-year survivals worldwide after heart transplantation are 81% and 65%, whereas here they are currently 94% and 80%.

According to the most recent statistics of the United Network for Organ Sharing (UNOS) that coordinates the provision of donor organs to recognized transplant teams and monitors their results, the average one-year survival following orthotopic liver transplantation in the USA is currently reported to be 76%. The liver transplant program at this institution, with a one-year survival of 91% (Table 2), is therefore already obtaining outstanding results when compared with the national average. This program has not been active long enough to allow longer survival rates to be compared with national figures.

Solid organ transplantation currently plays a major and steadily increasing role in the care of patients with end-stage single-organ disease. It is a demanding form of therapy that requires a high degree of expertise and dedication from the surgical, medical, nursing, and paramedical personnel (from multiple disciplines) involved in its delivery. Excellent survival results can be obtained by highly trained teams. These procedures offer new life with excellent quality to appropriately selected patients. Increasing consideration is being given to patients who require transplantation of more than one organ.

The major factor limiting a wider application of this form of therapy is the number of organ donors that become available. In light of the excellent survival statistics and high quality of life experienced following successful solid organ transplantation, every effort should be made to increase both organ donation and the referral of potential organ recipients to qualified transplant teams.

Acknowledgments

The authors gratefully acknowledge the very many members of the medical, nursing, and paramedical staff who have contributed to the care of the patients who have undergone organ transplantation at Baptist Medical Center. In particular, we recognize the major contributions of Drs. John Chaffin and David Van Hooser, cardiothoracic surgeons.

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The Authors

All authors are members of the staff of the Oklahoma Transplantation Institute at Baptist Medical Center in Oklahoma City.

David Cooper, MD, PhD, is a cardiothoracic transplant surgeon, director of research and education, and scientist-in-residence.

E.N. Scott Samara, MD, is a urologist and kidney transplant surgeon and director of the Kidney Transplant Program.

Luis Mieles, MD, is director of the Abdominal Organ Transplant Program.

Hadar Merhav, MD, is an abdominal organ transplant surgeon.

Norman Imes, MD, is a pulmonologist and co-director of the Lung Transplant Program.

David Van Thiel, MD, is a hepatologist and director of Transplant Medicine.

Nazih Zuhdi, MD, is director and chief transplant surgeon.

Creating More Generalists: Physician Reform by Reason ...or by Mandate?

DOUGLAS P. BEALL, MD

In a recent Gallup poll, 85% of those questioned felt that the choice of doctor was the most important item to retain in a new health care system. While the element of freedom of choice remains strong in the health care reform arena for people who receive care, it seems to have been relegated to a secondary concern for those individuals who provide care. For example, legislation has been introduced that calls for the physician workforce to be comprised of 50% primary care and 50% specialists prior to the turn of the century. This legislative proposal has been combined with a suggestion to limit the number of physician residency positions to the number of U.S. medical school graduates plus 10%. While these recommendations have been touted as a solution to many health care delivery shortcomings, no substantial indications exist to support these claims. Furthermore, when examined more closely, these suggestions appear to be increasingly contradictory.

The Counsel on Graduate Medical Education (COGME), a federal body created to advise Congress on physician supply, recommended (in its third report) the 50/50 division between specialists and generalists and the residency limitation of 110% mentioned previously. In their fourth report, however, the COGME admitted that "experimental data do not exist to define the proportion of generalists and specialists needed to provide optimal access to primary care services." Taking this into consideration, a logical question would be—if there is no data supporting the theory that an increase in generalists (now approximately 35% of all physicians) will increase the access to primary care, what is the rationale behind these recommendations? The answer lies deeper in the COGME's fourth report, where they cite the experiences of "other Western nations" such as Canada and Great Britain, noting that 50% of Canadian and 70% of British doctors are generalists. This hardly seems comforting considering the fact that numerous hospitals in Ontario were closed during the month of December due to lack of funding, and the British government is in the process of dismantling its National Health Service because it can no longer provide quality service at an acceptable cost.

The COGME's use of "other Western nations" as a pattern for health care reform seems somewhat unusual considering Western Europeans are now discussing how to shrink their cradle-to-grave welfare state and drastically reduce their health care expenditures. A recent report by independent analysts even predicted that within seven years, 1 British hospital in 3 will be forced to close or merge due to pressures to reduce costs.

Proponents for increasing the number of generalists have insisted that equalizing the specialist-to-generalist ratio will reduce costs without affecting the quality of health care. The intrinsic assumptions for this point of

The COGME admitted that "experimental data do not exist to define the proportion of generalists and specialists needed to provide optimal access to primary care services."

Direct correspondence to Douglas P. Beall, MD, 110 Centre Avenue A, Baltimore, MD 21222.

view are that specialists provide care at a higher cost and that specialists only treat what falls within their area of focus. In addition to the fact that the latter viewpoint has never received much credibility, recent studies indicate that once the higher severity of illness often seen in specialists' practices is controlled for, specialist care may actually be lower in cost for certain disease conditions.

To remove or limit the fundamental freedom of specialty choice would limit the possibility for any kind of rewarding experience for a large number of doctors.

In addition to efficient organization and cost control, the concept of physician career choice is an important component to workforce reform and one that has rarely been discussed by the popular media. Training requirements for physicians are among the most stringent and time consuming of any profession. Completion of all prerequisites commonly takes 10 years or more after college before a doctor can become board certified to practice in his area of expertise. The mandate for rapid change toward primary care would not only require that highly trained specialists undergo further generalist training but that a quota be made for medical schools to assure at least a 50% generalist graduation rate. The investment of time for physicians is an exercise in delayed gratification at best, and to remove or limit the fundamental freedom of specialty choice would limit the possibility for any kind of rewarding experience for a large number of doctors. What other profession is threatened by a governmental mandate of this type? Are lawyers mandated to practice a certain kind of law? Are teachers separated by quotas to teach a specific curriculum? Not only does the intrinsic nature of this approach fail to address the reasons why there is a lack of generalists, but it moves our health care training system further toward being centrally regulated as opposed to being regulated according to free market consumer demand. Ultimately this will give rise to a system that is neither rewarding for health care providers nor user friendly for health care consumers.

Finally, the proposal to limit the number of residency positions to the number of U.S. medical school graduates plus 10% is an idea that is much more complicated than its proponents would admit. Currently, the number of residency positions is approximately 120% of the number of U.S. medical school graduates. The additional 20% comprises a mixture of foreign medical graduates, individuals completing a second residency, and various other training scenarios. To reduce the number of positions to 110%, some residency programs would be limited in numbers of residents and others would be eliminated altogether. The proposal regarding the method of limitation suggests that all residency programs should be examined and ranked with the lowest ranking programs to be subsequently reduced in size or eliminated. Obviously, it is extremely difficult to apply an accurate ranking to any residency program given the lack of objective criteria and wide variety of training environments involved. Furthermore, the residency programs that tend to be ranked highest are those that are associated with prestigious academic centers and those located in large population centers. Logic dictates that if these types of programs are ranked highest, the residency programs that could be reduced or eliminated would be those that are located in rural or poverty- stricken inner city areas. These potentially expendable training programs, not surprisingly, happen to be comprised of a much greater percentage of primary care physicians than their higher ranking counterparts.

The dichotomy of the COGME's recommendations is therefore obvious, with unilateral mandates attempting to increase the number of generalists on one hand and a policy which will (in practical application) decrease the number of generalists on the other. Additionally, the proposed governmental control over the career choices and the actual practice of physicians is an unprecedented usurpation of personal and professional freedoms.

The purpose of this discussion is neither to dispel the notion that more generalists are needed nor to discredit the practice of primary care medicine. The evidence indicates, however, that if these policies are enacted for the purposes of increasing access to primary care, reducing costs, and producing more generalist physicians, the fundamental premise behind these formulations is flawed and needs to be reassessed. In short, not nearly enough pragmatism has been applied to adequately reshape a system that will affect nearly every American during the course of his or her lifetime.

The Author

Douglas P. Beall, MD, lived in Oklahoma City and was the OSMA's resident physician delegate to the AMA when he wrote this article. He now lives in Baltimore, Md.

The Politics of Medicine: Getting the Prescription Right

By REPRESENTATIVE DON MCCORKELL

This brief article, reprinted from the OSMA's July 1994 Legislative Summary, gives physicians an inside look at the art of political medicine.

DURING THIS YEAR'S legislative session dozens of critical health care issues were debated and decided ranging from prescriptive authority for optometrists to proposals requiring PPOs to compensate "any willing provider." State legislatures have always been the center of battle for health care debates. That is not going to change in the foreseeable future. In spite of recent attention at the federal level to proposals under consideration for universal health care, public policy on health care will continue to be substantially affected by state policy.

In such an environment you would expect doctors to be deeply involved in state politics—particularly state legislative politics—but such is not the case. Although doctors are represented by highly competent and professional lobbyists, they are themselves largely absent from the scene. Even the political influence of DOs, which only a few years ago was an awesome sight to behold, has faded dramatically. When the House of Representatives considered the bill granting prescriptive authority to optometrists, the gallery was filled with optometrists. The medical association's lobbyists were working religiously, but there was not an MD or DO to be found in the building. The optometrists not only won, they overwhelmed the opposition. What happened and why?

The following is a diagnosis and a prescription:

1. The medical profession is far less proactive and far more reactive than it should be in politics. Reactive politics is like acute care as opposed to preventive care—far greater risks are involved. In politics reactive actions are even more likely to be defined as driven by self interests than proactive actions. Look at the recent history of osteopathic doctors. When they were building their profession, they were actively engaged with legislators, persuading them that their proposals would enhance the quality of care for citizens. The optometrists have been especially politically active. They make sure that every legislator has at least one or more optometrists who know them personally and work closely with them. On a personal level, when someone you don't know tells you that you can't trust someone you do know and like, your natural reaction is skepticism at best—especially if you have limited expertise and have to base your decision on who you believe you can trust.

2. A lot of doctors, like a lot of businessmen, don't understand the constitutional principle of "separation of powers." A legislature is far more than a mildly active "board of directors." They set policy—not the governor. This is even more true in Oklahoma. Yet look where doctors and businessmen invest in politics in Oklahoma—they are busy trying to elect a "chief executive," thinking he is the CEO that will turn the company around, while they too often ignore the center of power in most democracies—certainly in this one—the legislative branch. (By the way, if we did not have a system of "separation of powers" we would probably be governed by the English and European parliamentary system—which is even more legislatively dominant since the legislature selects the executive)

3. Eternal vigilance is the price of liberty and democracy. Winning a battle one day does not win it forever, or even tomorrow. Losing it does not mean it is lost tomorrow. The Legislature is not "unresponsive." It is hyper-responsive and its memory is short. A doctor who participated in winning some major victories a few years ago, but who has been politically inactive in the interim, will face a rude awakening if he suddenly jumps into the fray to react to some new political proposal he finds distasteful. First, the same people aren't there anymore. Legislative turnover is very high (about 20% plus every two years.) The process is far more accelerated, with less time to devote to complex issues (ala short session reforms.) If he is lucky, he will get a couple of minutes to state his case. Meanwhile his opponents, who knew what they wanted to achieve, have been working for months, getting to know legislators, working in their campaigns, gently sharing with them the reasonableness of their position. The doc is going to go home and collapse in his easy chair as

Politics of Medicine *(cont.)*

frustrated like the former athlete who, having failed to play or exercise in ten years, suddenly runs out for some Saturday football on a hot day and is shocked by his injuries and heat exhaustion.

4. Politics is a participatory sport—not a spectator sport. The spectators are never happy in a participatory sport. They either go on the field and start playing, or they quit looking.

Doctors can have an enormous impact on health care policy and politics, if they want to. Here's the prescription (Remember: follow the instructions—don't take all the pills in the bottle at one time; don't try to do a year's worth of exercise on one day):

1. Get involved in politics. Get to know legislators and candidates on a personal, professional, and political basis. Don't make the mistake of limiting yourself to "your legislator" or "your party." If even one MD lives in my district, I don't know it—and I doubt it. If it takes 51 votes to win and the party of your affiliation has 30, you can't get there without another 21. It is simple arithmetic.

2. Make citizenship a habit, like brushing your teeth. Don't just make contacts; establish relationships.

3. Don't be discouraged by failures and setbacks—it happens to everyone. I have not only lost on issues, I have lost elections; but I found that "Never give up" is the most important advice and eventually leads to success.

4. Build alliances; identify others who should share your concerns and enlist their help.

5. Listen to the advice of your professional representatives—you hired them because of their skill, just as you would hire a specialist. When the specialist tells you that you have to be a part of the treatment, listen to his advice. Don't look for a specialist who says he can "fix" your condition without any effort on your part. That advice only comes from the charlatans in this business, just like it only comes from the charlatans in medicine.

6. Understand that just because someone disagrees with you, that does not make them your permanent enemy. The most effective people I know in politics are those who know how disarming it is for someone to come up to you, right after you voted against them, and express their understanding and desire to work with you in the future. Decision making in government is difficult, stressful, and challenging. There have been times that I have voted against my best friends because I thought they were wrong on a particular issue. The ones who were really friends were still friends after those votes.

This prescription will make a difference—but it is up to you to follow it.

Rep. McCarkell has served in the Oklahoma House of Representatives for sixteen years and is the senior member of the Tulsa County House Delegation. He has been an active and vocal member of the health committee for several sessions. Rep. McCarkell used his strong voice both in committee and on the House floor in opposition to SB 818, which allows optometrists to prescribe dangerous drugs.

Letters

A rose is a rose is... coercion?

To the Editor: I read your editorial ["Tricycle or Bicycle?" Sept. '94] with great interest. Please note that the word is coercion as to what is superimposed on a member by the State Medical Society in order to keep a "unified" membership.

This issue has nothing to do with the value of the society, which I certainly do not question for a moment, or its ability to function, or the need for the society. It has to do with coercion and making people join in order that they can get their PLICO Insurance without any difficulty.

I particularly bring your attention to the recent issue of the AMA *News* with the large cover on it. This stated quite clearly that the AMA has in fact agreed to one of Congress's Health Care proposals. This is a perfect example of why many of us get furious at the AMA.

On a personal basis, I realize that you have nothing but the very best motivation toward all of us in medicine and hence this is not to be considered a personal attack on you but on the system of coercion.

—Ernest G. Warner, MD
Oklahoma City

If you've ever wanted to be appointed to a state board...

The State of Oklahoma has hundreds of agencies, boards, and commissions. Oklahoma citizens, interested in serving the state in a volunteer capacity, comprise the membership of these boards and commissions.

Often, the Oklahoma State Medical Association is asked to provide the names of nominees to serve on these various boards and commissions. Some nominations are forwarded directly to the Governor; others are given to the Speaker of the House of Representatives or the President Pro Tempore of the Senate.

The OSMA is now compiling a list of individuals who would be willing to serve on one of these state boards or commissions. If you are interested in offering your services, please complete the form (both sides) provided on the facing page and send it, along with a brief biography or C.V., to: OSMA, 601 Northwest Expressway, Oklahoma City, OK 73118. Indicate on the form if you have an interest in a particular agency, board, or commission.

Please note that volunteers' names will go to a wide variety of boards and commissions, not just those dealing with medical issues. The OSMA believes it is important that community leaders such as physicians and their spouses not limit their volunteer service strictly to medically related entities.

EXECUTIVE APPOINTMENT APPLICATION

(The information asked for will be used to determine nominee qualifications and/or demographic accounting.)

Full Legal Name:

_____ _____ _____
Last First Middle

Home Address:

_____

_____

_____

_____
City State County Zip

Mailing Address (if different from above):

_____

_____
City State County Zip

Occupation:

_____

Employer:

_____

_____
Employer Address

_____
City State Zip

Home Phone __(_____)_____

Business Phone__(_____)_____

Fax Number____(_____)_____

Are you currently serving on any statewide Boards, Commissions, Task Forces, etc.? Yes No

_____
Term Expires

_____
Term Expires

Areas of Interest (name specific Boards, Commissions, etc. if possible)

_____

_____

_____

Demographics

Are you a United States Citizen? Yes No
Are you registered to vote in Oklahoma? Yes No
How long have you been a resident of Oklahoma? _____Yrs _____Months
Do you have any relatives who are employees of the State of Oklahoma? Yes No

_____
Name Agency

_____
Name Agency

List below any professional affiliations

_____

_____

_____

***** The following questions are voluntary. They are not used in consideration for appointment unless statutorily required. However, this information is important, so please answer if applicable. *****

Gender - Male Female Race _____

Date of Birth _____ Religious Affiliation _____

What is your political affiliation? Democrat Republican Independent Other _____

Have you ever been convicted of a felony? Yes No
(If yes, please explain)

_____
College of Graduation Year

_____
Medical School Year

Please return this completed form to: OSMA
Mr. David Bickham
601 Northwest Expressway
Oklahoma City, OK 73118

or Fax completed information to: (405) 842-1834

Contact David Bickham at the OSMA headquarters (405) 843-9571, if you have any questions.

COMMENTS:_____

_____

_____

HEALTH DEPARTMENT

Coccidiomycosis follows the Northridge, Calif., earthquake

Since the January 17 earthquake in Northridge, California, the number of cases of coccidiomycosis reported in Ventura County (adjacent to Los Angeles) has dramatically increased; although only 52 cases were reported from this county in all of 1993, 170 persons were diagnosed between January 24 and March 15, 1994. The Centers for Disease Control and Prevention (CDC) is conducting an investigation to determine the possible association between the earthquake and this outbreak.

It is now known that over 50% of persons being infected with this dimorphic fungus have no symptoms; most of the remainder develop acute influenza-like illness characterized by fever, chills, and cough. In most people these symptoms resolve spontaneously; only very rarely, in less than 1% of cases, does disseminated disease occur. Around 40% of those infected develop symptoms of the primary infection between one and four weeks after exposure, but dissemination may be insidious, with recognizable symptoms occurring years after the primary infection. Disseminated disease is more common in African-Americans, Filipinos, other Asians, pregnant women, and patients with AIDS or other forms of immunosuppression.

Coccidioides immitis, which causes coccidiomycosis, grows in the soil of the Southwest, from California to Southern Texas, which explains some of its other names: San Joaquin Fever, Valley Fever, and Desert Fever.

Most cases of coccidiomycosis occur in people living in or traveling through the Southwest, but infection can occur after contact with dusty fomites from the endemic areas. Although this disease can affect anyone, more than half of the patients are between 15 and 25 years of age and males are much more likely to have disease than females.

Any persons having lived in or traveled through the Southwest, and in particular those who have been to Ventura County since the Northridge earthquake, should be considered at risk for developing coccidiomycosis. For further information about this disease contact the Acute Disease Service of the Oklahoma State Department of Health at 405/271-3266.

WORTH REPEATING

The following article appeared in THE WALL STREET JOURNAL, *issue of 9/6/94.*

HMO Juggernaut

U.S. Healthcare Cuts Costs, Grows Rapidly And Irks Some Doctors

A Few Patients Are Slighted, Squeezed Specialists Say; But Firm Also Gets Praise

How Katie Avoids Hospital

By RON WINSLOW
Staff Reporter of THE WALL STREET JOURNAL

BLUE BELL, Pa.—A joke making the rounds in Philadelphia-area doctors' lounges goes like this:

Leonard Abramson, chief executive officer of U.S. Healthcare Inc., the big health-maintenance company, dies and goes to heaven, where he tells God what a great place it is. "Don't get too comfortable," God advises. "You're only approved for a three-day stay."

That's a kind of cost control that the messianic Mr. Abramson understands. In the past two years, U.S. Healthcare has slashed the fees it pays to specialists and hospitals by 12% to 20% and sometimes more, these providers say. In the past year, it has cut members' days in hospitals by 11%. Increasingly, it ask specialists and hospitals to assume the financial risk for procedures that cost more than anticipated.

Tight Cost Controls

U.S. Healthcare is widely considered one of the country's toughest HMO companies, and one of the most innovative. It reins in medical costs so tightly that it keeps 30 cents of every premium dollar to pay for salaries, marketing, administration and shareholder dividends, nearly 10 cents more than the industry average. It zealously tracks the performance of doctors and hospitals, paying more to those whose quality scores are high. It is earning robust profits—up to 99% in the past 24 months—while rocking the tradition-bound healthcare markets along the East Coast.

"Unless you change the culture of the community you're working in, you're not changing health care," Mr. Abramson declares.

However, the company's hardball tactics have made some enemies, particularly among specialist physicians, whose income and power are most threatened by its success. They contend that the cost savings, rather than benefiting patients or society, are simply diverted from providers to its stockholders and managers, notably Mr. Abramson.

The Pay Issue

Last year, the 61-year-old entrepreneur, who once drove a cab to put himself through pharmacy school and who founded the company 18 years ago, received $9.8 million in salary, bonus and stock options. His dividends on his 15 million Class B shares run $11.4 million a year.

David F. Simon, the company's principal legal officer, says Mr. Abramson's "compensation is definitely not excessive. The company's shareholders as well as its CEO are deserving of the rewards that come in a free-market economy."

But the controversy over U.S. Healthcare goes far beyond Mr. Abramson's pay. As HMOs gain members and power, the company is on the leading edge

HMO Juggernaut *(continued)*

of the changes sweeping health care. Managed care is shifting the focus from high-tech specialty treatment to primary care and prevention. HMOs are holding doctors and hospitals accountable for quality while paying them under incentives to employ fewer, not more, medical procedures.

More Gains Likely

Whether this is quality medicine or just cheap medicine stirs fierce debate even as prospects for managed care grow stronger. Some analysts estimate that $20 billion to $30 billion in medical-care dollars are up for grabs in the metropolitan New York market alone as HMOs finally penetrate one of the last bastions of traditional, fee-for-service medicine. Employers continue to steer employees to HMOs while new government initiatives are opening up Medicare and Medicaid populations to managed care.

"We stand on the threshold of a new era in which managed-care plans will probably dominate" health care, Arnold S. Relman, former editor of the *New England Journal of Medicine,* wrote in a recent issue. "Yet there is much we do not know about the consequences of this revolution."

Many doctors complain that U.S. Healthcare, and its managed-care competitors, select hospitals for their networks based on discounts, not quality; hinder patients' access to specialists; and deliberately cater to generally healthy people who need fewer medical services. They also contend they are induced to make decisions based on what's good for the HMO rather than what's good for the patient.

"If I send too many patients to the dermatologist, I get penalized through no fault of mine, "says Rajendra Prasad, an internist and gastroenterologist in Trenton, N.J. "If I save money for the HMO, I get rewarded. It puts the patient at odds with the doctor."

U.S. Healthcare says its cutbacks in fees to specialists and hospitals aren't as deep as its critics contend, though it acknowledges that its drive to reduce unnecessary procedures and hospital stays does pare providers' incomes. Mr. Simon adds: "The company's primary objective has always been to improve the quality of care. When you improve quality, you naturally reduce the costs of that care."

U.S. Healthcare is a managed-care juggernaut with 1993 revenue of $2.6 billion. Its membership of 1.7 million is growing at a better-than-10% annual clip. Its seven HMOs have contracts with a total of more than 6,000 primary-care doctors, 24,000 specialists and 300 hospitals.

"Just a few years ago, many providers were hoping they could get away without having to do business with them," one hospital official says. "Now, they are so powerful, that's no longer an option."

In its core markets in Pennsylvania, New Jersey and New York, U.S. Healthcare enjoys commanding shares of HMO

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Toll Free 1-800-522-9219

enrollments. One reason: Its charges are generally well below traditional health-insurance premiums and competitive with other managed-care plans.

But like other HMO companies, U.S. Healthcare faces challenges. Growing competition, customers' rising bargaining power and more regulatory pressure helped limit U.S. Healthcare's average increases in premiums this year to less than 3%, after a 12% rise last year; premiums could be flat in 1995. In addition, some analysts think the company has extracted almost all the savings it can get from doctors and hospitals.

"To get quality, you can't keep chopping away at fees. You've got to pay up," says Kimberly Purvis, an analyst at Donaldson, Lufkin & Jenrette Securities Corp.

So, future growth depends heavily on U.S. Healthcare's ability to attract new members while continuing to manage the costs and delivery of care. Its chief agents of change are primary-care doctors. While it has antagonized specialists, it cultivates its ties with the internists, pediatricians and family doctors who deliver routine care to its members and control their access to specialists and hospitals.

The Compensation System

Like many other HMO companies, U.S. Healthcare pays these doctors a monthly fee for each patient, a strategy known as capitation. Unlike most HMOs, it also pays a bonus—up to 28%—based on how the doctor meets various quality and customer-service targets.

A doctor or practice gets added compensation, for instance, for scheduling office hours at night, for linking up with the HMO by computer and for accepting and retaining new U.S. Healthcare patients. High immunization rates for children and mammography exams for women over age 40 are rewarded. Doctors whose patients spend fewer days in hospitals or make fewer visits to specialists are paid more. But the company says it makes sure patients aren't denied needed care; it even can dock the pay of physicians whose referrals to specialists seem low.

In evaluating doctors, U.S. Healthcare audits more than 100,000 medical charts a year and sends doctors monthly reports detailing how often their patients use hospitals and specialists, for instance, or how they rate on patient satisfaction—and comparing them with other doctors in the network. It also mails more than a million surveys to members, whose views influence their doctors' pay.

That evaluation system is a boon to doctors such as Theodore Robinson, who heads a six-doctor pediatric practice in suburban Philadelphia. He says the bonus he and his partners get lifts their compensation for U.S. Healthcare patients above that from patients paying on a per-visit basis. Their 2,800 U.S. Healthcare members amount to about 20% of their patients but account for about one-third of their annual revenue, Dr. Robinson says.

Many doctors are reluctant to admit that financial incentives are necessary to promote good medical practice. But U.S. Healthcare says its doctors show consistent annual im-

(continued)

In Memoriam

1993

William Finis Ewing, Jr., MD ..... March 31
John Robert McInnis, MD ..... March 31
Chester Randall Seba, MD ..... April 4
Edward Cotter Murray, MD ..... April 9
Eric McLain White, MD ..... April 23
John Franklin DeJarnette, Jr., MD ..... May 1
Harold Alwin Smith, MD ..... May 4
Kenneth Warren Bonham, MD ..... May 28
John Raymond Stacy, MD ..... July 6
Carl Davis Osborn, MD ..... July 11
Mark Royal Johnson, MD ..... July 12
W. Turner Bynum, MD ..... July 13
James Dougherty Martin, MD ..... July 31
Edward David Greenberger, MD ..... August 6
Horace Harold Porter, MD ..... August 15
Clifford A. Traverse, MD ..... August 20
J. Wendall Mercer, MD ..... September 2
Emma Jean Anthis, MD ..... September 27
Russell D. Harris, MD ..... September 28
Walter Lee Honska, Jr., MD ..... October 9
Ethel Maurice Walker Heras, MD ..... October 25
James Burton Pitts, Jr., MD ..... October 29
John Daniel Capehart, MD ..... November 20
Kevin James Cammack, MD ..... December 4
William Best Thompson, MD ..... December 10
Henry Grady Ryan II, MD ..... December 17

1994

Fannie Lou Leney Hayward, MD ..... January 2
Kirk Thornton Mosley, MD ..... January 3
Richard Charles Wade, MD ..... January 6
Austin Walsh Webb Haddox, MD ..... January 13
Earl Mathiews Woodson, MD ..... February 20
Tom Lamar Johnson, MD ..... March 5
Orville Main Rippy, MD ..... March 11
Minor Elliott Gordon, MD ..... March 14
George Loren Norris, MD ..... March 27
Max A. Glaze, MD ..... April 29
Winfred Aaron Showman, MD ..... May 14
Mark Daniel Holcomb, MD ..... June 1
Carter William Mathews, MD ..... June 3
Frank Wilson Clark, MD ..... June 6
Harold Ray Sanders, MD ..... June 15
Robert Bruce Howard, MD ..... June 16
Richard Warren Loy, MD ..... July 7
John Hobson Veazey, MD ..... July 11
Wesley A. Whittlesey, MD ..... July 12
Laurence Sevier McAlister, MD ..... July 19
Jon Meyer Chenette, MD ..... August 4
Beryl Drew Henwood, MD ..... August 14
Jess Duval Herrmann, MD ..... August 16

HMO Juggernaut *(continued)*

provement in such measures as immunization rates, cholesterol testing and keeping medical charts.

Money helps. When the company recently offered 1,200 doctors a free course intended to improve breast-cancer screenings, just 150 showed up. Then it dispatched a team of employees to recruit doctors for the course, promising the recruiters a $50 bonus for each physician who attended. Another 550 doctors came. Determined to do better, the company added the course to its compensation plan, essentially paying doctors about $400 to take it. "We got 95% of them," says Arthur N. Leibowitz, a medical director. "There's a study there on how you get physicians to do things: You pay them."

To track care givers' performance, the company relies on its U.S. Quality Algorithms unit. With a desktop computer, Steven Zatz, the unit's president, can rank hospitals according to infection rates of urology patients, by the length of stay for coronary-bypass surgery or by the number of babies delivered by Caesarean. "We can go to a hospital and say, `This is how your performance looks compared to the best hospitals in the region,'" Dr. Zatz says.

Few hospitals have such information on their own performance, much less know how they stack up against competitors. "Hospitals are ceding power and authority to managed-care companies because we can't control our own information," says an official of one hospital that has contracts with U.S. Healthcare. "They have the data; we don't."

Increasingly, the company is using that data to negotiate the same kind of performance-based contracts with hospitals that it has with primary-care doctors, agreeing to pay a bonus for low rates of infection or second operations, shortening hospital stays and meeting other standards.

Targeting Specialists

And it expects to bring specialists, still generally paid according to a fee schedule, into a quality-compensation plan as well. Over time, that is likely to significantly reduce the number of specialists in its networks. Already, officials say, about 80% of referrals go to about 20% of its specialists, and Dr. Zatz's data will help it identify those offering high quality and low costs.

Another way U.S. Healthcare says it improves quality is through programs to manage chronic diseases such as diabetes, asthma and congestive heart failure that often put patients in the hospital when symptoms get out of control.

After an asthma attack hospitalized seven-year-old Katie Henry for five days last Christmas, the company provided equipment that enables her parents to monitor and treat her at home before she has a full-fledged attack. "Before, by the time we knew she was having a bout, there was nothing we could do but go to the emergency room," says Brian Henry, her father.

Since then, Katie hasn't been to the hospital, she has missed less school, and her parents have missed less work taking care of her. Katie's pediatrician, Michael B. Torrence, benefits, too, because the program helps raise his quality rating.

"Managed care isn't just doing something that's cheaper," Dr. Torrence says. "It's going to the store and getting your money's worth."

All of this helps explain, among other things, why all six U.S. Healthcare HMOs that have been evaluated by the National Committee for Quality Assurance, in Washington, have won full, three-year accreditation, the best performance of any U.S. managed-care company.

Of course, there are skeptics. One is Kenneth Peacock, an orthopedist in Mount Laurel, N.J., who says he has participated in meetings to develop protocols for using magnetic resonance imaging and other technologies in diagnosing and treating orthopedic patients.

Working for Less Money

"I left those meetings thinking all we did was figure out ways for U.S. Healthcare to save money," Dr. Peacock says. He thinks he has already done that: The company pays him about $475 for a wrist procedure known as carpal tunnel release, he says, nearly $1,000 less than his customary charge and as least $150 less than other managed-care companies pay.

The discontent in New Jersey has provoked a backlash among some physicians and hospital executives, many of whom see HMOs in a conflict of interest between their members and their shareholders. Some 3,500 doctors, mostly specialists, recently invested $5,000

DEATHS

Frank Wilson Clark, MD
1917 - 1994

Frank W. Clark, MD, a 1951 graduate of the University of Oklahoma Medical School, died June 6, 1994, in Ardmore. Dr. Clark was born in Celina, Tenn. He established his surgical practice in Ardmore in 1955, after completing his postgraduate work in McKinney, Tex.. He retired in 1987 after more than 30 years in practice and was named an OSMA Life Member the same year.

Beryl Drew Henwood, MD
1917 - 1994

OSMA Life Member Beryl D. Henwood, a native of Lipscomb, Tex., died August 14, 1994. Dr. Henwood joined the U.S. Navy after graduating from high school, serving in the Pacific during World War II. After leaving the Navy he completed his undergraduate work and went on to earn his medical degree from the University of Tennessee Medical School in Memphis in 1955. He began his family practice in Collinsville, Okla., in 1957, and remained there until his retirement in 1989. After closing his family practice, Dr. Henwood worked part-time with the Indian Health Bureau and various Indian Hospitals. In 1968, Dr. Henwood served as president of the Tulsa County Chapter of the American Academy of General Practice.

each—a total of $17.5 million—to help launch Physician Healthcare Plan of New Jersey.

In a similar effort, First Option Health Plan of New Jersey is seeking to raise capital from hospitals. Dr. Peacock wants to organize a group of orthopedists to market services to managed-care companies. "I want to sit down with the biggest stick I have when it comes down to negotiating for a slice of pie," he says.

Henry Rosin, a surgeon and president of Physicians Healthcare Plan, says U.S. Healthcare and other managed-care companies compromise quality by intimidating doctors into withholding care. His group contends that it can deliver higher-quality care at competitive prices while enabling doctors to keep more of the proceeds. "To have stock in [U.S. Healthcare] is wonderful—it's a highflier," Dr. Rosin says. (Since 1988, the stock is up twelvefold.) "But if one is going to revamp the entire American health-care system, the answer is not in making a lot of money for shareholders."

Such fledgling efforts face enormous hurdles in raising the capital and developing the information systems needed to get a foothold in the market. U.S. Healthcare says it doesn't feel threatened. Mr. Simon maintains that such doctors are concerned first with their own pocketbooks and that they, not U.S. Healthcare, face a conflict of interest. "If you're both the provider as well as the HMO, there's an inherent conflict of interest," he says.

But while some providers talk of challenging managed-care companies, many are giving in. Despite misgivings about U.S. Healthcare, Thomas Gabuzda, chief of hematology at Lankenau Hospital in Philadelphia and president of the Philadelphia Medical Society, says he recently signed contracts with the company and another HMO. The reason: His hospital, after long resisting managed care, had signed up. Some longtime patients joined HMOs, too.

As for its profits, U.S. Healthcare offers no apology. The company says it fills a need by improving quality, reducing costs and, in the process saving employers and consumers hundreds of millions of dollars. Educational Testing Service, of Princeton, N.J., says its health-care premiums are 25% to 30% lower for employees who are U.S. Healthcare members than for those in its traditional insurance plan.

Uwe Reinhardt, a Princeton University economist, likens HMO operators to 19th century bounty hunters who reaped big rewards for rounding up criminals that sheriffs couldn't catch. Nowadays, the bounty is waste in the health-care system. Not trusting government to root it out, Americans have engaged HMOs to do the job, Prof. Reinhardt says. He adds that Mr. Abramson shouldn't be criticized for becoming rich by providing "adequate health care at a reasonable price. He's doing exactly what we're asking him to."

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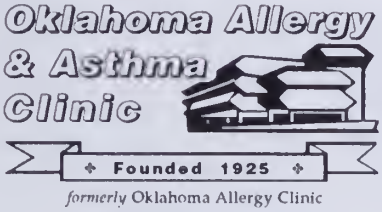

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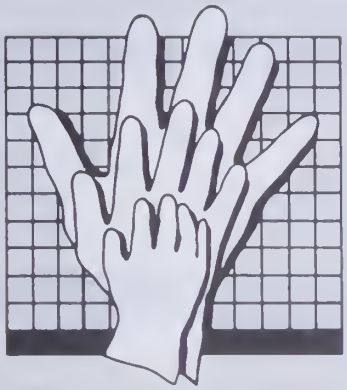

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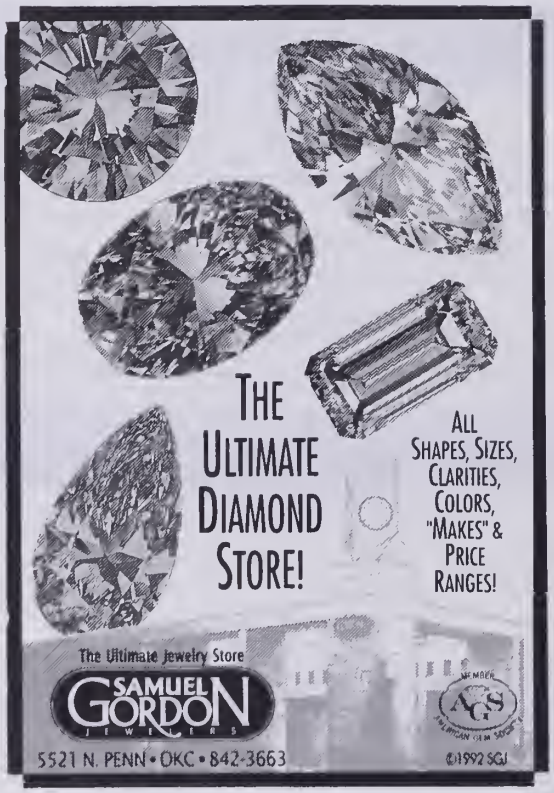

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I will if you will!

November's here... the elections are behind us... hope all of your candidates won! And even more than that... I hope you were out there helping them win. They'll remember you a little easier when you ask them to vote your way. With the '94 election hoopla out of the way, the real work is here to be done!

Those issues we warded off last year will more than likely reappear in the 45th session of our state legislature. And we must be ready to fight the good fight! To be really successful, every physician and spouse in Oklahoma needs to become proactive in the legislative process... our OSMA staff works hard on our behalf, but we are the constituents, the ones electing those lawmakers and the ones who have their ear. I hope you all had the opportunity to read "The Politics of Medicine: Getting the Prescription Right" by Representative Don McCorkell as it was printed in the July, 1994, Legislative Summary. It prescribed what all of us in the medical community need to be doing. If you haven't read that article, contact the OSMA or me, and I will see that you get it. [The article is reprinted on page 511.]

Health system reform will again be the discussion of choice at the state and in our nation's capital and it's imperative that we be present in the discussions. Statewise, we will probably again see the prescriptive authority bills. We lost the round to the optometrists last year but were successful in defeating the bills introduced by the chiropractors and advanced practice nurses. Senator Angela Monson's Health Care for Everyone bill died on the floor... but more than likely a similar bill will surface this year.

In Washington, President Clinton will continue to push for health system reform, and we must continue to interact with our Senators and Congressmen, letting them know the kind of reform that is necessary to make the system better... not to destroy the finest healthcare in the world.

One of the ways you can do this nationally is to join the P.O.W.E.R. Network (Physicians Organized to Work for Effective Reform) of the AMA, By joining, you will be kept informed and given the information to make you an effective player in the process. The toll-free P.O.W.E.R. Network Hotline number is 1-800-833-6354. By simply calling that number, you can hear a message describing Congressional and Administration activity, its impact on medicine, and clear instructions on how you can make a difference on important upcoming votes. The AMA is making every attempt to provide education about what is happening and to make it easy for you to interact. All you have to do is dial the number!

Another great educational tool is *American Medical News*. Physicians, read it... and then take it home for your spouse to read. You will learn what is happening nationally as well as what other states are doing.

One way to make a difference in what is happening in the state legislative arena is to become a legislative contact. Establish a relationship with your state legislator so that he or she looks forward to hearing from you when legislation dealing with medicine is on the agenda. *You* have the expertise that is necessary to help that legislator cast a knowledgeable vote. With thousands of bills introduced each session, they need all the help they can get... and I assure you, the allied health professions are well represented. On your day off, take your spouse and spend a day at the state capitol visiting with your legislators and watching a little legislation happen. Believe me, it will make you a believer in your being involved in the process! Claudia Kamas, our OSMA lobbyist, has worked to establish legislative contacts in all of our districts and your willingness to take an active part will be welcomed by her. Just contact her at the OSMA, (405) 843-9571 or 1-800-522-9452.

As legislation crucial to organized medicine moves through the system, we must often make immediate contacts with our legislators; thus we must quickly get the message out to physicians and spouses to make those calls or contacts. One way to accomplish this is through the legislative contact system, but another way is through the use of phone banks or telephone trees. The county alliances are very effective at getting this done, as many already have in place county telephone calling committees to get information out to their members, and could easily activate those networks when the need arises for quick contact with our legislators. I strongly urge all counties to have this network in place so that you can be effective when time is short. And, doctor, be available to take the call from the alliance member and then make your call to your legislator. Yours may be the one telephone call that makes a difference in how your legislator votes!

Make the commitment today to become involved and do your part to see that our state and national governments make the right choices when it comes to health system reform. Speak out and let your voice be heard as a positive voice for medicine. All of us together... physicians and spouses... must be the watchdogs of government to ensure that patients continue to receive the best healthcare in the world. I challenge you today to call or send a card to your legislators at the state and national levels and let them know that you are available with "an expert opinion" and that you plan to be involved when issues affecting medicine are on the table! Let's not let it be said again that doctors were missing from the scene when medical issues were debated. Let's be there front and center! I will... if you will!

—Sherry S. Strebel, Chair
Legislative Affairs

■ **With a large contingent of graduates in Oklahoma,** Washington University School of Medicine, St. Louis has provided the following information about some of its upcoming CME courses: (1) Women's Healthcare Issues, Nov. 18, 1994, Ritz-Carlton Hotel, St. Louis; (2) Gateway to Exploration: 12th World Congress of Endourology and ESWL and 10th Basic Research Symposium, Dec. 2-6, 1994, Adam's Mark Hotel, St. Louis; and (3) Cardiology for the Geriatric Patient, Dec. 10, 1994, Ritz-Carlton Hotel, St. Louis. For information on any of these seminars, contact the Office of Continuing Medical Education, Washington University School of Medicine, Campus Box 8063, 660 S. Euclid Ave., St. Louis, MO 63110-1093; phone 1-800-325-9862, FAX (314) 362-1087.

■ **"Complications and Controversies in Obstetrics and** Gynecology" is the title of the 29th Southwestern Gynecologic Assembly Meeting to be held December 1-3, 1994, in Dallas. Co-sponsoring is the University of Texas Southwestern Medical Center at Dallas, which designates this continuing medical education activity for 12 AMA credit hours. ACOG has assigned 13 cognate hours. Registration fee is $325. For information, contact J. Patrick McCarty, MD, Registration Chairman, Southwestern Gynecologic Assembly, 7777 Forest Lane, Bldg., C #234, Dallas, TX 75230, (214) 661-7711.

■ **Copper Mountain, Colo., once again is scheduled to** host the Irwin Brown Memorial Physicians' Winter Retreat. This will be the 22nd consecutive year for the retreat, scheduled for December 26, 1994–January 2, 1995. For registration information contact Dr. Warren M. Crosby, Department of Obstetrics and Gynecology, University of Oklahoma Health Sciences Center, P.O. Box 26901, 4SP700, Oklahoma City, OK 73190, 405-271-7449.

■ **Dr. G. Lance Miller, Tulsa, immediate past president** of the OSMA, has been appointed to the Oklahoma Breast Cancer Prevention and Treatment Advisory Committee.

■ **February 1, 1995 is the deadline for nominations for** the Ayerst Wyeth Physician Award for Community Service and the Donald J. Blair Friend of Medicine Award. The service award goes to a physician who, in addition to his or her medical practice, has contributed significantly to the community. The Blair award, formerly the Layman of the Year Award, is presented to a non-physician for his or her support of the art and science of medicine in Oklahoma. Winners are selected by the OSMA Board of Trustees at their February meeting each year and the awards are presented at the Annual Meeting of the OSMA House of Delegates in April. Winners in 1994 were Bertha M. Levy, MD, Oklahoma City, and Wilson D. Steen, PhD, Norman.

Nominations for both awards should be sent with supporting material to Douglas C. Hubner, MD, Chairman, OSMA Board of Trustees, 601 Northwest Expressway, Oklahoma City, OK 73118.

■ **The boards of PROklahoma Care, a physician-owned** and -operated MCO, and the Oklahoma Physician Network, an IPA, approved the bylaws, stock prospectus, and other paperwork at their October 12 meeting, paving the way for the marketing of their stock, expected to begin by early December. Once officially chartered, the two corporations will be governed by their respective boards, both of which include representatives from the Oklahoma Osteopathic Association.

OSMA members of the PROklahoma board are Drs. Donald Crawley, Terry Dolan, Eldon Gibson, Joe Hester, James Jones, Perry Lambird, Larry Long, Mary Anne McCaffree, William McCurdy, G. Lance Miller, David Selby, R.B. Winters, Ken Whittington, and Mike Winzenread. OOA members are Drs. Richard Langerman, Terence E. Grewe, and Ronnie B. Martin.

Members of the board of the Oklahoma Physicians Network, are, from the OSMA, Drs. Jon Axton, Richard Boatsman, Ed Calhoon, Carl Critchfield, Billy Dale Dotter, Jay Gregory, Douglas Hubner, Carol Blackwell Imes, Billy Irons, Nick Knutson, Ron Kreger, Dennis McIntyre, William Morris, Tim Smalley, Bruce Storms, and Boyd Whitlock, and from the OOA, Drs. Joseph R. Schlecht, Sheila G. Simpson, and Gordon P. Laird.

While the stock offering for PROklahoma Care will include explanatory material, board members and the OSMA staff believe that personal visits from them are critical to the success of the program. County medical society presidents, specialty society presidents, and hospital medical chiefs of staff are urged to schedule a PROklahoma Care presentation as soon as possible. To do so, contact OSMA Executive Director David Bickham or Associate Director Mike Sulzycki, 405-843-9571 or 1-800-522-9452.

■ **All PLICO-insured physicians in Oklahoma are required** to attend a loss prevention seminar every three years or face losing their professional liability coverage. The last of the 1994 PLICO seminars was in September, but physicians still needing attendance credit for this year have one option remaining: viewing a videotape program and completing a self-administered test afterwards. Cost of the program is $100. Physicians wishing to exercise this option should contact Debbie Thurmond at the OSMA, 405-843-9571 or 1-800-522-9452.

■ **"Through the Looking Glass: Percutaneous Evaluation** and Treatment of Spinal Disease" is the title of the 31st Annual Orthopaedic Symposium to be presented next spring by St. Luke's Episcopal Hospital in Houston. The two-day meeting, April 28-29, 1995, wil be at the Doubletree Hotel Post Oak in Houston. For details call 713-791-4200.

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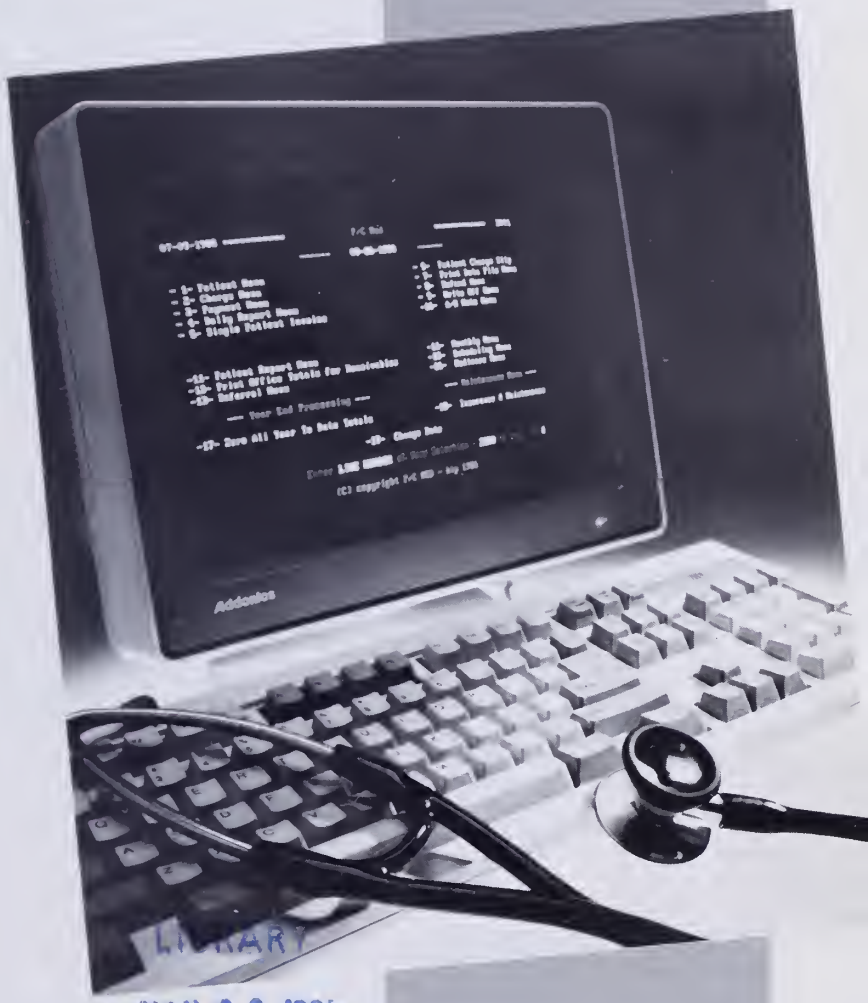

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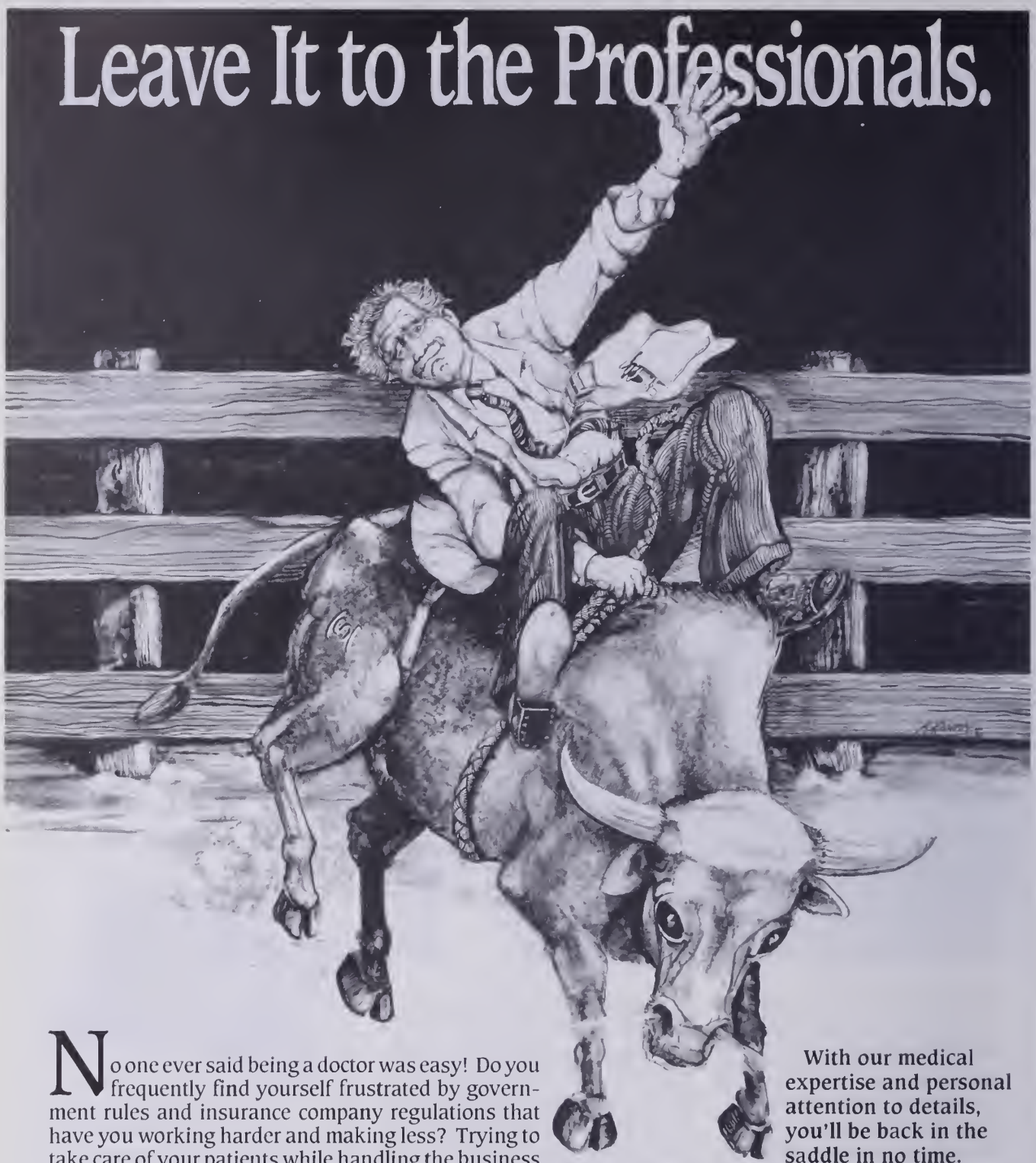

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The JOURNAL OF THE OKLAHOMA STATE MEDICAL ASSOCIATION (ISSN 0030-1876) (USPS 285-000) is the official publication of the Oklahoma State Medical Association and is published monthly under the direction of the OSMA Board of Trustees at 601 Northwest Expressway, Oklahoma City, OK 73118, (405) 843-9571. Second Class postage paid at Oklahoma City, OK 73125.

POSTMASTER: Send address changes to JOURNAL, c/o Oklahoma State Medical Association, 601 Northwest Expressway, Oklahoma City, OK 73118.

Subscription to the JOURNAL is included in membership dues. All other subscriptions are $30 per year. Single copies are $3 per copy, prepaid, subject to availability.

Reprints of articles are available from the authors or from University Microfilms International, 300 North Zeeb Road, Dept. P.R., Ann Arbor, MI 48106, 1-800-521-3044.

National advertising representative: State Medical Publishers Network, 9534 Marshall Drive, Shawnee Mission, KS 66215-1354, (913) 888-3758, 1-800-788-2360,

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Official Publication Of the OSMA Since 1908

JOURNAL

OKLAHOMA STATE MEDICAL ASSOCIATION

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Who Needs Ethics?

Once upon a time, ethical behavior was held in very high regard by most physicians. A generation ago, when a more personal patient contract was common, most people sincerely believed that physicians, and the medical profession generally, represented the highest order of ethics to be exhibited in American society.

For many years the Gallup Poll organization has periodically interviewed a random sample of American adults and ranked the various occupations by the Interviewees' opinion of "the honesty and ethical standards" of those practicing the occupations. Formerly, medical doctors consistently ranked at the very top of the ratings list, usually with clergymen and pharmacists being the principle competitors for top billing.

But the latest Gallup Poll ranking of the occupations, compiled from September 1994 interviews and printed in local newspapers, places medical doctors well down the rankings list, with five other occupations rated to be higher in ethical behavior. On reading the list, it is a questionable consolation to find that car salesmen, Congressmen, and lawyers remain below physicians in ranking.

The poll now gives top ranking to occupations formerly rated further down the list, and the trend disclosed by the pollsters appears to be valid. The direction of the change is troubling to conscientious physicians, as it signals that patient confidence in the ethics and honesty of our profession is now markedly lower.

Whence commeth this downfall for the medical doctors of America? Why are today's physicians less respected than their antecedents? Medical school admission criteria remain high. Academic standards still cull the unfit. Physicians' clinical effectiveness has been increased by starting technical advances, and is the greatest ever and still increasing.

The reason for this "fall" may be uncertain, but—except for the technical improvements—the most drastic change in medical practice has been the wholesale insertion of third party payment mechanisms into our medical economics. Almost every patient now expects third party intervention. Nowadays we physicians all too often are as beholden to the government agency and the insurance contract as we are to the patient, and thus we can no longer look the patient in the eye and honestly say. "This course I believe to be the best for you." And the patients sense this infidelity and no longer consider their physician to be as honest as their druggist and clergyman.

If this is to be the era of reform in health care delivery, let us reform the third party payor system, and re-direct the flow of funds through the hands of the patients, so that we physicians will again keenly feel our responsibilities to that special human being who is a patient.

Human patients heal better when they are treated by trusted physicians with whom there is rapport. The presence of a warm and trusting relationship shortens many illnesses, and soothes many patients' dissatisfactions. We physicians need to re-establish our integrity and veracity with the people we serve.

We all need ethics.

Ray V. McIntyre, M.D.

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Ho! Ho! Ho!

You better watch out, you better not cry, you better not pout, I'm telling you why...

Oh, how those words and the refrains from other very familiar Christmas carols echo through my head during this time of year. For a few moments, if you would, close the door to your office, have your receptionist hold all calls, and recall with me memories from your past Christmases.

Remember when we were children and actually believed in Santa Claus? Do you recall hearing those bells and someone shouting *Ho! Ho! Ho!* either on Christmas Eve or morning? Remember how you sprang to your feet and ran into the living room, or den, or fireplace area where the tree was—or in my case to the front porch since we didn't have a chimney—and there to your amazement you found that special toy you had been dreaming about for the past several months? Oh, the thrill and excitement of that moment! How magical that instant when you believed that there really was a Santa Claus and that he came to your house to leave you this very special gift. What a gift it would be to be able to relive that moment and really experience those feelings again.

Remember the years passing and our coming to the realization that there really wasn't a fat little guy in a red suit riding in a sleigh being pulled by flying reindeer. But still, at that special moment when we heard the bells and the *Ho! Ho! Ho!*, we sprang to our feet and rushed to that special place to find, once again, that special item that only our parents or grandparents knew we wanted. And, for just a second or two, remember how magical that moment was: it was as if there really were an entity that cared enough to deliver that special item.

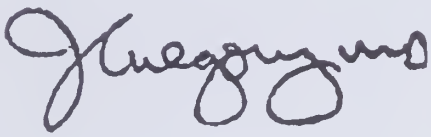
Remember how the years have passed and how we have now become the ringers of the bells and the shouters of *Ho! Ho! Ho!* in an attempt to recreate that magical moment for our children. Now we are the ones who stay up until midnight or one A.M. wrapping presents, stuffing the turkey, and putting together that bicycle or doll house in the frigid garage while following instructions that read "easy assembly; no tools required." Oh, to go back and relive the memories of childhood.

All of us know the true meaning of Christmas regardless of our faiths or beliefs. We have heard that Christ was born on this day, and that he came to earth to give us everlasting life if we only believe. It's all there in the Bible. You know the story by heart if you are a believer. If you're not, it does make for interesting reading.

During this holiday season, I'm going to challenge each and every one of you to do something to create in those around you the belief that you truly are someone special. I challenge each of you to go out every day during this time of year and commit a senseless, random act of kindness. By holding the door open for a stranger; by picking up a half dozen angels from the angel tree in the mall; by saying *please* and *thank you;* by being kind, caring, and compassionate to your patients, family, and colleagues; by delivering a couple of bags of groceries to someone who is hungry; or by emptying your closet of those items of clothing you haven't worn for the past two years so that someone may have warm new clothing this time of year, you will be saying to others by your deeds that they, too, are someone special and that you really care. Every day commit a senseless, random act of kindness; it really will make a difference and, who knows, it may become a habit!

Have a very joyous holiday season and remember to recreate that magical moment for those around us.

Your friend,



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Gastrointestinal Problems Experienced in an Intensive Care Unit

Harlan I. Wright, MD; Abdul Nadir, MD; Paolo Caraceni, MD; Farah Naz-Nadir, MD; Nicola DeMaria, MD; David H. Van Thiel, MD

Medical emergencies involving the gastrointestinal tract include bleeding, abdominal pain, diarrhea, toxic megacolon, pancreatitis, and hepatic failure. Each can be a cause for admission to an intensive care unit or occur de novo in a patient hospitalized in such a unit. The clinical presentation, management, and consequences of these varied gastrointestinal problems seen in an intensive care unit are presented followed by a recommended course of action.

The intensive care unit (ICU) represents the single site in the hospital where the most severely ill patients are concentrated and where physicians from multiple specialties and subspecialties often share in the care of a single patient. With the proliferation of subspecialty intensive care units, the types of problems seen in a given unit frequently reflect the subspecialty focus of the unit. Thus, the problems seen in a coronary care unit are different from those seen in a respiratory care unit or a burn unit and differ markedly from those seen in a neurosurgical care unit. Nonetheless, certain problems remain as common problems that are seen frequently in an intensive care unit irrespective of its subspecialty focus. Many of these later problems are gastrointestinal problems. Moreover, units that are departmental in focus, such as medical or surgical intensive care units, appear to experience a broader spectrum of gastrointestinal problems than do subspecialty ICUs. It is the clinical recognition, awareness of risk factors, and management of the gastrointestinal problems seen in ICUs that is the focus of this review.

From the Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, Oklahoma City, Oklahoma 73112.

Direct correspondence to David H. Van Thiel, MD, Oklahoma Transplantation Institute, Baptist Medical Center of Oklahoma, 3300 Northwest Expressway, Oklahoma City, Oklahoma 73112.

Gastrointestinal Bleeding

Over one quarter of a million people are admitted to hospitals annually in the United States for acute gastrointestinal bleeding.[1] The three most common sources of gastrointestinal bleeding are acute gastritis, peptic ulceration, and esophageal variceal bleeding.[2] In general, the mortality rate for gastrointestinal bleeding has remained rather stable at 10% over the last quarter century with individuals who are older than 60 years of age, those with other organ systems that are dysfunctional, and those who experience rebleeding within a 48-hour period having the poorest prognosis. Such cases account for greater than 90% of the cases that ultimately will die during their hospitalization which has been either initiated or complicated by the bleeding episode.[3] Table 1 shows the frequency of gastrointestinal bleeding noted in 233 patients submitted to a general medical intensive care unit for whom immediate or at least near immediate (within 6 hours of admission) upper gastrointestinal endoscopy was performed.[1]

In general, the mortality rate for gastrointestinal bleeding has remained rather stable at 10% over the last quarter century

As can be seen from the table, gastric bleeding due to gastritis or gastric and/or prepyloric ulceration are the most common sites of bleeding and account for 48% of all cases. The second most common site of upper gastrointestinal bleed-

Table 1. Endoscopically Identified Bleeding Sites in Consecutive Patients Admitted to a Medical ICU for Gastrointestinal Bleeding

Site	Frequency
Esophagitis	3%
Esophageal varices	10%
Gastritis	22%
Peptic ulcer	46%
Gastric	14%
Prepyloric	12%
Duodenal	20%
No bleeding site identified	19%
Total	100%

ing is the duodenum (22%) with the esophagus being the least (13%) likely site for gastrointestinal bleeding seen in a non-subspecialty intensive care unit. Despite early endoscopy, the origin of 19% of the cases of acute upper gastrointestinal bleeding are never identified.[1] When one sees a patient with gastrointestinal bleeding, several questions need to be addressed immediately. These are: How much bleeding has occurred? At what rate has the bleeding occurred, and from what site? The answers to these important questions are readily obtainable from the immediate history and a rapid examination of the patient. Postural changes in the vital signs suggest that a blood loss equivalent to or greater than 20% of one's blood volume has occurred. Shock is present when the blood loss is greater than or equal to 40% of an individual's original blood volume. The latter can be calculated to be 5% of the individual's body weight when measured in kilograms.

The presence or absence of urine flow is a major prognostic sign as well, and the absence of urine production suggests major bleeding, either in terms of its overall volume or rate. Conversely, the hematocrit is a most unreliable measure of either the rate or severity of acute bleeding until at least 48 to 72 hours have elapsed from the time of the bleed and equilibration has had time to occur.

Historical clues as to the site of bleeding include the presence or absence of melena, hematemesis, hematochezia, abdominal pain, or a history of either prior gastrointestinal bleeding or hepatic disease. Table 2 provides a listing of the historical clues which the authors have found useful for determining the site of bleeding when a patient is evaluated initially.

Stress Gastritis.— Bleeding that occurs de novo after a patient has been hospitalized, particularly in an intensive care unit, is much more likely to be of gastric origin and to be due to "stress gastritis."[4] Often over-looked sites, however, are the esophagus (particularly those patients with nasogastric or nasoenteric tubes) or rectal varices (limited to patients with pre-existing advanced liver disease).

Stress gastritis, a diagnosis that is often made endoscopically, occurs in 60% to 100% of cases endoscoped in an intensive care unit.[4] Fortunately, stress gastritis that presents clinically as bleeding is seen much less often and occurs in only 5% of cases seen in an intensive care unit. Moreover, in the last decade, with the development of better methods of resuscitation and the introduction of stress gastritis prophylaxis with any of a number of accepted regimens (hourly administration of antacids; H_2 blockers; omeprazole; sucralfate or early enteral feeding), the incidence of clinically important stress gastritis has declined substantially.[5] The use of antacids as a prophylaxis for stress bleeding can lead to the milk-alkaline syndrome in those with co-existent renal dysfunction, diarrhea in those using magnesium-based antacids, or constipation or an antacid bezoar in those utilizing aluminum-based antacids.

The term *stress ulceration* is a misnomer, as it rarely, if ever, consists of overt ulceration. Rather, stress gastritis typically consists of intramucosal hemorrhage (petechiae and ecchymosis) and a wet, dusky, or cyanotic mucosa that occasionally also demonstrates erosions. These findings are usually localized to the body of the stomach.[6] Not all stress lesions are identical, however. Those associated with major burns (Curling's ulcers) are true ulcers that can perforate. Unlike classical stress gastritis, which is not associated with an increased acid secretory rate, this unique form of stress bleeding is associated with excessive gastric acid secretion. Despite these differences in the pathophysiologic and etiologic characteristics of varying types of stress bleeding,

Table 2. Clues to the Site of Gastrointestinal Bleeding

	Upper	Lower
History	Peptic ulcer disease Hematemesis	Diverticulitis Hematochezia
N-G Aspirate	Bloody	Clear*
BUN	Increased	Normal

*Must have bile

gastric acid secretion and its control are important in the clinical management of all types of acute gastric bleeding.

Table 3 shows the consequences of neutralization of secreted acid upon bleeding rates and the factors that are thought to control or reduce bleeding and especially rebleeding rates.[7] As is indicated in the table, as gastric pH is progressively increased, the frequency of bleeding, its severity, and the risk of rebleeding is reduced substantially.[7,8] Therefore, the institution of some form of gastric acid neutralization or secretion inhibition should be initiated in all patients hospitalized in an intensive care unit who are at risk for gastrointestinal bleeding. These include patients with major body burns, head injury or coma, those having experienced major trauma or multiple abdominal surgical procedures, those in shock or manifesting evidence of sepsis, those with major organ failure (lungs, kidney, or liver), and those with a recent history of alcohol, aspirin or NSAID usage.[8]

Even with intensive therapy directed at either neutralization or inhibition of gastric acid secretion, patients with myocardial infarction, respiratory insufficiency, renal failure, multiple trauma, or sepsis will experience gastrointestinal and particularly gastric mucosal bleeding in 5% to 20% of cases.[9] Risk factors for bleeding which have been identified in such cases include longer durations of hospitalization within the ICU (>7 days), the need for mechanical ventilation, especially if such need persists for >5 days, and the presence of renal failure necessitating dialysis. Because the surgical mortality of stress gastritis is substantial (30% to 50% of cases) and consists of a total gastrectomy, if it is to be successful, medical therapy and especially stress gastritis prophylaxis is crucially important. The data available addressing the issue of stress bleeding in an ICU are best for those with stress gastritis occurring in patients with major body burns.[10] Eighty-six percent of patients with burns involving >35% of the body will experience an episode of acute gastrointestinal mucosal hemorrhage.[10] In greater than three-fourths of these cases, the site of the bleeding will be found to be in the stomach. Gastric and duodenal erosions occurring in the same individual are seen in 72% of such cases. Gastric or duodenal perforation occurs in 6% of such cases. Surgical intervention for bleeding or a complication of bleeding is required in 13% of cases.

Factors that can be controlled or managed, in addition to gastric acid secretion and gastric pH, which can reduce the incidence of stress bleeding are shown in Table 4. The correction of each of these factors has been shown to reduce the rate of stress bleeding in ICU bound patients in one or more studies.[3,5,8,9]

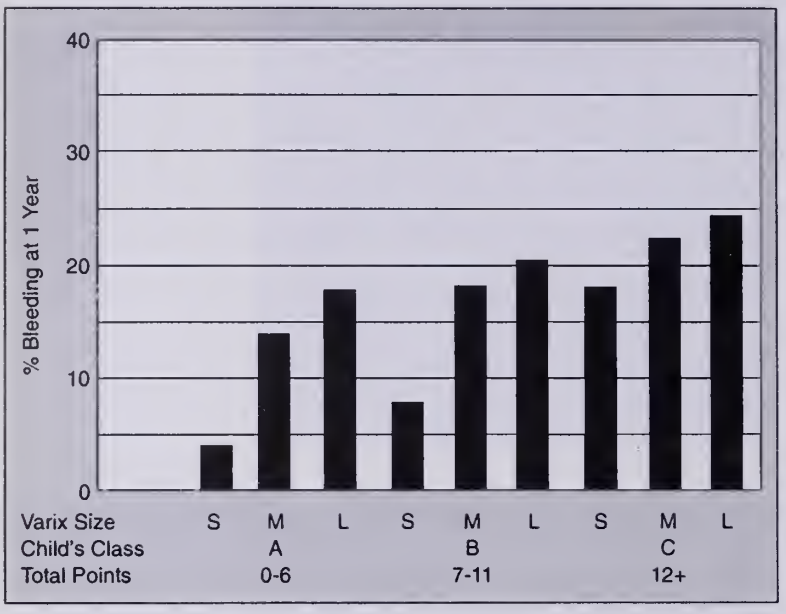
Table 3. Gastric pH, GI Bleeding and Coagulation

pH	Result
>3.5	Decreased bleeding rates
>4.5	Inactivation of pepsin
>5.0	99.5% of acid neutralized and blood clotting can occur
>5.5	Improved coagulation and platelet function
>7.0	Reduced rebleeding rates
>8.0	Pepsin is destroyed

Table 4. Factors That Modify Gastrointestinal Bleeding Sites in Patients in a Medical ICU

Respiratory Factors	PAO_2 Hemoglobin level Cardiac output Oxygen consumption Cardiac output distribution
Hepatic Factors	Splanchnic shunting Congestive gastropathy Coagulopathy Hepatic function Hypersplenism
Renal Factors	Acid-base balance Azotemia
Volume Factors	Ischemia Critical organ hypoperfusion Dilutional coagulopathy
Nutritional Factors	Enteral feeding

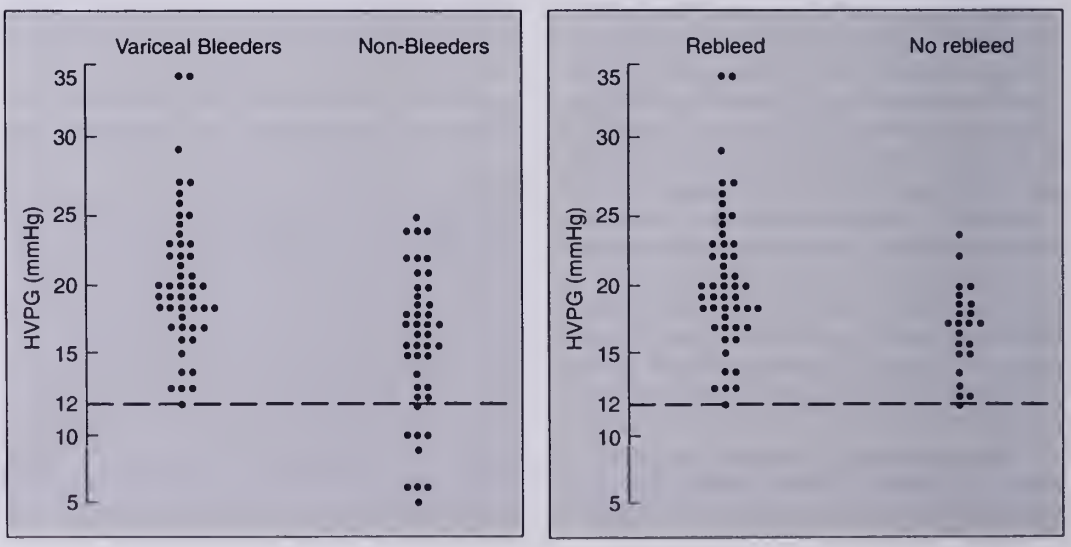
Peptic Ulcer Disease.— Acute bleeding due to peptic ulcer disease is more common from a duodenal ulcer than from a gastric ulcer. Nonetheless, the clinical management of ulcer disease, regardless of its location, is nearly identical. Acutely, the major goals should be to identify the site of bleeding and to stop it endoscopically using either mucosal injection with saline, alcohol, or norepinephrine, the use of a heater probe, or electrocoagulation. After the bleeding has stopped, measures directed at controlling acid



secretion become important. Surgery is indicated in cases that occur in older individuals (those over age 60 years), those who bleed rapidly (>5 units per 24 hours) and those who experience prolonged or repetitive bleeding (>20 units and more than one episode of bleeding).[11] The surgical management of such cases is considerably better than that associated with stress bleeding and typically necessitates vagotomy with pyloroplasty or a super selective vagotomy and, less often, a subtotal gastrectomy. The latter surgical procedure is usually reserved for elective surgical procedures. The utilization of early endoscopy and either heater probe or injection methods of bleeding control had reduced the number of cases that in the past went to surgery. Moreover, endoscopy often identifies those patients who ultimately will require surgery and allows special attention to be given to them (those with active bleeding or visible vessels) prior to their actual need for a surgical procedure.

Esophageal Varices.— Esophageal variceal bleeding is a major untoward consequence of advanced hepatic disease. As such, it often precipitates a state of hepatic decompensation that leads directly to the patient's death. Because of its importance as a risk factor for death, considerable attention has been given to the problem of bleeding varices, the ability to predict in whom it is likely to occur and, most importantly, its treatment and prevention. Figure 1 graphically shows the inter-relationships between variceal size (small, medium, and large), the Child's-Pugh classification, and the risk of variceal bleeding. This clinical information, plus the fact that bleeding rarely occurs when the hepatic venous portal vein gradient (HVPG) is <12 mmHg enables the endoscopist to identify high risk patients for the application of additional therapies such as variceal sclerosis, banding, or a TIPS (transjugular intrahepatic portosystemic shunt) procedure.[12] Figures 2A and 2B show the cumulative risk of bleeding and the risk of surviving based upon a single parameter, the HVPG.[12] The important thing to note is that these two figures are nearly identical in their shape and for their critical cut-off values ED 50 value for each. Data such as these are particularly valuable in performing clinical studies and enable the randomization of patients having similar disease severity into the various arms of a clinical study.

Whenever acute variceal bleeding is consid-



Figures 2A and 2B.

ered a possibility, the patient should be admitted to an intensive care unit. This is followed with or occurs simultaneously with vascular resuscitation and the institution of acute pharmacotherapy with either vasopressin plus nitroglycerin or, preferably, an intravenous bolus and subsequent infusion of somatostatin analogue (octreotide) to either curtail acute bleeding and/or prevent rebleeding. Both agents are effective and can be instituted immediately and utilized until emergency endoscopy can be performed to recognize, treat, or exclude variceal bleeding as the source of the bleeding episode. Because of the cardiovascular complications associated with the use of vasopressin, the use of octreotide has been gaining in popularity for this particular use.

Emergency sclerotherapy is the initial therapy of choice. It can reduce early deaths and forestall subsequent rebleeding episodes.[15] Sclerotherapy is not without risk, however. The untoward consequences of emergency sclerotherapy and their frequency are shown in Table 5. The frequency of these complications appears to be a function of the experience of the sclerotherapist more than of the technique or agent being used by the sclerotherapist.[15]

The major decision to be made for patients with variceal bleeding is what one should do if emergency sclerotherapy does not control the bleeding. The available options include the use of a Sengstaken tube, more aggressive or repeat variceal sclerosis, esophageal transection, portal venous decompression, an emergency TIPS procedure or liver transplantation. The choices between these various options are determined by their availability locally and the expertise of the physicians and surgeons available to perform them. The authors prefer the TIPS procedure as a bridge to liver transplantation or a distal splenorenal shunt.

Portal hypertensive gastropathy remains a clinical problem, and often gets worse after the immediate problem of variceal bleeding is controlled. In fact, bleeding following sclerotherapy control of varices is often not a result of recurrent variceal bleeding but rather a consequence of gastric mucosal bleeding occurring as a result of portal hypertensive gastropathy. This particular lesion involves the proximal stomach and is characterized by vascular ectasia, mucosal hemorrhage, and a peculiar mosaic pattern of the gastric mucosa.[16] The mucosa also often appears rather cyanotic, wet, and friable. Fortunately, the treatment of portal hypertensive gastropathy is identical to the pharmacologic measures used to control or prevent recurrent variceal bleeding (eg., ß blockade). The addition of surface protective agents such as sucralfate or prostaglandin E_2 may enhance the prophylaxis for bleeding experienced with ß blockers in patients with severe portal hypertensive gastropathy, although this has not been proven.

Table 5. Efficacy, Type, and Frequency of Sclerotherapy Complications

Efficacy		
	Control of acute bleed	86-95%
	Esophageal variceal obliteration by 3 months	83-90%
	Mortality within 6 weeks	25-38%
Complications		
Problem	**Rate Per Sclerotperapy Session**	**Rate Per Patient**
Ulceration	19.5%	40%
Esophageal leak	1.4%	7%
Esophageal stricture	2.6%	13%
Esophageal rupture	0.3%	1.6%

Lower Gastrointestinal Bleeding.— Lower gastrointestinal bleeding is much less likely to occur acutely than is upper gastrointestinal bleeding and is, as a result, less often seen de novo in an intensive care unit. The primary causes of lower gastrointestinal bleeding that are recognized as being a cause of acute bleeding occur either as a result of diverticular bleeding (common), bleeding from inflammatory bowel disease (less common), ischemic bowel disease involving the colon which often occurs in older individuals hospitalized for medical reasons other than gastrointestinal bleeding and occasionally rectal varices or hemorrhoidal bleeding in patients with portal hypertension.

Diverticular bleeding typically presents as acute bleeding episodes which are painless and associated with no other abdominal signs or symptoms.[17] The diagnosis is typically made by exclusion but often is made after the identification of colonic diverticula. Right-sided diverticula appear to bleed more commonly than do left-sided diverticula. When bleeding is massive or occurs repetitively in an older individual, either a right hemicolectomy or a total colectomy with rectal sparing is the treatment of choice, after initial stabilization of the patient. A specific bleeding site or location can occasionally be identified using either nuclear medicine bleeding

Probably the most frequent gastrointestinal complication seen in any ICU is antibiotic-associated diarrhea due to pseudomembranous colitis.

scans or angiography. When such a site is identified, a lesser rather than greater surgical procedure is indicated. With the use of aggressive or emergency colonoscopy, more cases of left-sided diverticular bleeding or confirmed right-sided diverticular bleeding are being recognized, and more local resections are being performed for diverticular bleeding than was possible in the past.

Ischemic colitis typically complicates some other major medical problem usually related to an underlying cardiovascular or atherosclerotic disease process.[18] However, it can occur in any situation in which intestinal hypoperfusion occurs, such as in shock or sepsis, or as a result of embolic disease. Typically, ischemic colitis presents with abdominal pain or intestinal cramps followed in one to two hours with bloody diarrhea.[18] Often, the abdominal pain is disproportionately great as compared to the patient's physical findings. This combination of findings ought to suggest the diagnosis of ischemic colitis, particularly in an elderly individual. The therapy of ischemic colitis should be directed at correcting the underlying pathology (sepsis, shock, poor cardiac output) if at all possible. If transmural necrosis occurs, it will become evident as a result of the development of peritoneal signs, an increasing leukocytosis and progressive hemoconcentration. In such cases, resection of the necrotic bowel is required and typically is followed by a second-look operation 24 to 48 hours after the initial resection. In the absence of a transmural necrosis, therapy includes both vasodilatory and cardiotropic agents (dopamine plus dolbutamine) and volume expansion to reduce the hematocrit to a level between 30% and 35%. This should reduce the vascular viscosity, increase the cardiac output, and fill the available vascular volume. Focal colonic stricturing can occur 3 to 4 weeks to several months after an acute episode of ischemic colitis.

Bleeding from rectal varices and/or hemorrhoids in a patient with advanced liver disease complicated by portal hypertension occasionally can be mistaken for massive upper gastrointestinal bleeding because of the rapidity and intensity of the bleeding. Thus, any patient suspected of having upper gastrointestinal bleeding and advanced liver disease, who is found *not* to have upper gastrointestinal bleeding, should have a flexible sigmoidoscopy to exclude bleeding from a rectal site. When such bleeding is identified, either tamponade therapy using a large (30-50 cc). Foley catheter placed in the rectum, varix banding, or sclerotherapy using any of the commercially available agents for esophageal variceal sclerotherapy can be utilized to control the bleeding. In unusually severe cases a TIPS procedure may be necessary.

Massive colonic bleeding in cases of inflammatory bowel disease (IBD) is rare but is more common in cases of Crohn's colitis than it is in cases of ulcerative colitis.[19] Thus, partial colonic resection for bleeding is performed more often in patients with Crohn's disease than it is in patients with ulcerative colitis despite the fact that, overall, more colonic surgery is performed in patients with ulcerative colitis than for Crohn's colitis.

The principal therapy for bleeding due to inflammatory bowel disease is not surgery but rather blood and blood products. Both hemoconcentration and hypovolemia are to be avoided in patients with inflammatory bowel disease as venous thrombosis of intestinal and extraintestinal sites can occur with either hypovolemia or hemoconcentration because of the intrinsic hypercoagulability that is associated with inflammatory bowel disease.

A diagnosis of intestinal perforation can be quite difficult to make in a patient with IBD unless free air is evident either clinically or more often by abdominal radiography. Clinical signs of intestinal perforation include fever, abdominal pain, tenderness, and overt clinical toxicity.[20] When a colonic perforation occurs in a hospitalized patient with ulcerative colitis, it almost always occurs as a free perforation and is a result of toxic megacolon (two-thirds of all cases). Perforations typically occur with the first attack of toxic megacolon (>50% of the cases). In contrast, an intestinal perforation occurring in a patient with Crohn's disease rarely is a free perforation. Instead, it is usually a contained perforation that can become an abscess. Rather than involving the colon, intestinal perforations in Crohn's disease usually occur in the distal ileum or occur at a site immediately proximal to a distal site of obstruction. In cases occurring in ulcerative colitis, urgent surgical intervention with a total colectomy is indicated. In cases of intestinal perforation occurring in patients with Crohn's disease, prolonged intravenous antibiotic therapy is indicated; surgery can be delayed and typically is performed to relieve the distal obstruction primarily and only secondarily to remove or drain the contained perforation.

Toxic megacolon is defined as a paralytic ileus involving the colon, typically the transverse and right colon, resulting in distension of the colon to a diameter >10 cm associated with signs of clinical toxicity such as fever (>105°F), tachy-

cardia (>120/min), leucocytosis (>10.5 × 10^3 cells/mm^3), anemia (Hct <30%), dehydration, azotemia, and often hypotension.[21] Toxic megacolon is a dangerous condition which is complicated frequently by a free perforation, if colonic decompression or resection is not performed. The patient's age and the timing of the onset of the toxic megacolon in a given patient's disease history are important variables determining the ultimate prognosis. An age >46 years is associated with a 30% or greater mortality rate while an age <40 years is associated with a mortality rate of 5%.[20,21] Moreover, the earlier in the hospitalization that the toxic megacolon is treated, the lower the risk of a lethal outcome. The use of new immunosuppressive agents consisting of either cyclosporine A or FK 506 administered intravenously has reduced the need for urgent surgery in patients with toxic megacolon.

Whenever a decision is being made to consider surgical intervention for toxic megacolon, it is important to realize that, on average, 30% of the cases in whom surgery is not selected ultimately will require surgical intervention at a later date.[21,22] Thus, unless strong indications mitigate against surgical intervention after an initial 48 to 72 hours of medical therapy, if significant improvement in the patient's condition has not occurred, total colectomy is indicated.

Pseudomembranous Colitis

Probably the most frequent gastrointestinal complication seen in any intensive care unit is antibiotic-associated diarrhea due to pseudomembranous colitis. The syndrome of pseudomembranous colitis classically consists of fever, diarrhea, and abdominal cramps occurring in an individual who either is on or has recently been receiving antibiotic therapy.[23] Most cases can be considered to be a "benign" antibiotic-induced diarrhea and will resolve with discontinuation of the antibiotic regimen. In a minority of cases (10%), severe disease with pseudomembranes occurs (67% of the cases).[24] It is important to recognize that pseudomembranous colitis can occur in the small bowel as well as the large bowel, although the right hemicolon and cecum are the most frequently involved sites followed by the entire colon and left colon, respectively. In patients receiving opiates, anticholinergic agents, and anti-diarrheals, constipation rather than diarrhea can occur. In such cases the diagnosis can be made either as a result of an assay for *C. difficile* toxin, culture of *C. difficile* from the stool, or endoscopically.

Overgrowth with *C. difficile* and a toxic response to the B toxin of *C. difficile* is the cause of this disease. In rare cases, the A toxin of *C. difficile* may not be present in the stool. Because most RAI for *C. difficile* toxin utilize the A toxin as the test ligand, the test may be negative despite the presence of *C. difficile* and clinically overt disease produced by the B toxin.

Therapy for pseudo-membranous enterocolitis consists of oral vancomycin or metronidazole and, rarely, the intravenous administration of metronidazole in very severe cases. In very mild cases, the oral administration of cholestyramine, which absorbs the two toxins, often is sufficient therapy and antibiotics are not needed. The presence of endoscopically identifiable pseudomembranes mandates antibiotics rather than cholestyramine therapy.

Acute Pancreatitis

Acute pancreatitis is an acute inflammatory condition of the pancreas characterized by abdominal pain, fever, and leucocytosis. Biochemically it is associated with a mild to moderate hyperamylasemia and lipasemia. Typically, alcohol abuse produces acute relapsing cases of pancreatitis of moderate severity. In contrast, gallstones and drugs, other than ethanol, produce severe pancreatitis that often leads to one or more of the complications of acute pancreatitis including a pancreatic phlegmon; pseudocyst; abscess; acute gastrointestinal, intrapancreatic, or retroperitoneal hemorrhage; and intestinal ischemia. As a result of the landmark work of individuals like Ransom et al., the identification of more severe cases of pancreatitis associated with complications and/or death can be recognized either at onset or, at the latest, 48 hours after clinical recognition of this disease process.[25]

The facts of importance at the time of admission which portend a poor prognosis because of pancreatitis include age >55 years, a glucose level >200 mg/d, leucocytosis >16,000 cells/mm^3, a serum LDH level >700 iu/l, and a serum SGOT level >250 iu/l.[25] Additional factors that identify an individual as having a poor prognosis within the first 48 hours include a serum calcium level <8 mg/dl, a PAO_2 <65 mmHg, a base deficit >4 mEq/l, a BUN increase >5 mg/dl, a hematocrit drop >10%, and a fluid requirement >6l in order to maintain normal vital signs. Should 3 or less of these features be present, the mortality associated with acute pancreatitis is 3% or less, and the risk of serious disease is only 11%. If more than 3 are present, the risk for death is 62%. The usual cause of death in cases of pancreatitis is sepsis occurring in association with multiple organ fail-

Patients with fulminant hepatic failure should be hospitalized in an intensive care unit and have regular assessments of their liver size, neurologic status, blood sugar, and coagulation status.

Table 6. Etiology of Fulminant Hepatic Failure

Viral Hepatitis	60-75% cases
HAV	
HBV/HB+HD	
HCV	
HEV	
Herpes simplex virus	
CMV	
EBV	
Adenovirus	
Drugs	20% cases
Poisonings	5-10% cases
Vascular Conditions	
Budd-Chiari	
Portal vein thrombosis	
Traumatic vascular injury	
Veno occlusive disease	
Malignant Infiltration with Lymphoma	
Miscellaneous Rare Conditions	

ure. CT scanning with IV contrast often enables detection of focal areas of underperfusion (i.e., potential necrosis) within the pancreas. Such an investigation is particularly valuable for identifying areas for potential CT-guided skinny needle aspiration for culture and the selection of appropriate antibiotics in severe cases of pancreatitis. Moreover, these same areas are the most likely foci from which subsequent pseudocysts or abscesses will form in cases of complicated pancreatitis.

Fulminant Hepatic Failure

Fulminant hepatic failure is defined as the onset of hepatic failure complicated by coma which occurs within 8 weeks of the clinical onset and in the absence of a pre-existing liver disease.[26] Severe fulminant hepatic failure has the same definition but has a shorter time course (2 weeks). The major causes of fulminant hepatic failure are shown in Table 6. Patients with fulminant hepatic failure should be hospitalized in an intensive care unit and have regular assessments of their liver size, neurologic status, blood sugar, and coagulation status. In addition, a central venous line, endotracheal tube, nasogastric tube, and foley catheter should be inserted to maximize patient care and monitoring. The use of an extradural pressure device for monitoring intracranial pressure is currently an optional "research" tool.[26,27] Factors that predict survival in fulminant hepatic failure are the duration of the disease process, the maximum stage of encephalopathy achieved, the patient's age, the etiology of the fulminant hepatic failure, and the presence or absence of complications such as gastrointestinal bleeding, infection, and renal failure.[28]

Survival is reduced in individuals greater than age 40 and is markedly reduced, to essentially zero without transplantation, in those older than 60 years of age. Survival declines from a value of 66% in individuals with grade 1 encephalopathy to 48% in individuals with grade 2, a value of 10% in individuals with grade 3, and <5% individuals with grade 4 encephalopathy. Clinically relevant prognostic factors include acidosis (pH <6.3), a factor 5 level < than 20%, a serum bilirubin level >20 mg/d, a serum creatinine >3.0 mg/d, and an α-fetoprotein level <20 mg/ml.[28-31] The most frequent cause of death in fulminant hepatic failure is cerebral edema (40% to 80% of deaths) followed by gastrointestinal bleeding (30% to 70% of deaths) and finally, sepsis.[28,29] Clinical signs of cerebral edema in fulminant hepatic failure (FHF) include hyperventilation, pupillary dilation, increased muscle tone, decorticate posturing, and respiratory arrest. The precise mechanism(s) responsible for the cerebral edema in acute hepatitis encephalopathy is unclear, and both vasogenic and cytotoxic mechanisms have been postulated.[29,30] Liver transplantation is currently the procedure of choice for individuals who progress beyond grade 2 encephalopathy.[31] Transplantation results in a 65% to 70% survival which is 2- to 3-fold greater than that achieved with the best medical therapies for patients with the same degree of hepatic encephalopathy.[31]

Summary

The preceding has been a review of the clinical presentation and management strategies used to treat gastrointestinal problems experienced in an intensive care unit. It is anticipated that this review will be of value to physicians in practice, those in training, and particularly those who rotate on or serve in intensive care units.

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The Authors

Harlan I. Wright, MD, and Abdul Nadir, MD, are staff physicians at the Oklahoma Transplantation Institute (OTI), Baptist Medical Center of Oklahoma (BMCO), in Oklahoma City.

Paolo DeMaria, MD, and Nicola DeMaria, MD, are research fellows at the Oklahoma Medical Research Foundation (OMRF) and at BMCO.

Farah Naz-Nadir, MD, is also a research fellow at BMCO.

David H. Van Thiel, MD, is the medical director of transplantation at BMCO.

Congenital Thumb Hypoplasia

Ghazi M. Rayan, MD

Hypoplastic thumb is a short, underdeveloped thumb with deficient or absent intrinsic muscles with or without deficient extrinsic musculotendinous structures.

Thumb hypoplasia is a common congenital abnormality with five types of increasing severity. This abnormality may be associated with local musculoskeletal deficiencies or systemic diseases and syndromes. In severe cases, basic hand function, including pinch and grasp, may be adversely affected. A variety of operative reconstructive procedures can provide thumb stability, and improve motor control and overall hand function.

Embryology

Streeter[26] described a 23-stage system for upper limb development. The limb bud (Wolff's crest) appears on Day 24 after fertilization (4 mm crown-rump embryo).

The limb bud has a core of mesenchymal tissue covered by ectodermal layer. Located at the apex of the bud is the apical ectodermal ridge (AER) which is essential for the continued limb differentiation and growth. The hand plate appears on Day 33. The thumb and other digital rays become evident on Day 38, and they begin to separate on Day 40 by the programmed cellular death of the tissues between the digits. Digital separation is complete on Day 47, and on Day 52 the digital pulps appear. Programmed cellular death of the AER takes place, and the limb development is complete by Day 56 (28 mm crown-rump embryo). The period of hand and thumb development and differentiation is short and takes place between the 4th and 8th weeks after fertilization.

The lateral plate mesoderm gives rise to connective tissue, bones, tendons, ligaments, and limb vasculature. Limb musculature is derived from somitic (somites) mesoderm that migrates into the developing limb bud. These cells form the anlagen of the limb muscles and differentiate into myoblasts or muscle cell precursors. Muscles are not necessary for nerve development and muscle formation, and differentiation is not affected by neural influences.[6]

Etiology

Thumb aplasia most probably results from damage to the apical ectodermal ridge near the end of the 7th week following fertilization. Thumb hypoplasia with thenar muscle deficiency is probably due to disturbance of the migratory capacity of the myoblasts from the somitic mesoderm.

As in most congenital differences, thumb hypoplasia may be due to idiopathic, genetic, or teratogenic factors. Most frequently the etiology is multifactorial and due to a combination of the patient's genetic make-up and environmental factors such as trauma, local vascular insult, toxic agents, viruses, etc. Miura et al[19] studied a group of patients with syndactyly, cleft hand, and hypoplastic thumb. They observed a lack of similarity between the last two conditions and there-

From the Baptist Medical Center of Oklahoma and the University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma.

Direct correspondence to Ghazi M. Rayan, MD, Physicians Building D, 3366 Northwest Expressway, #700, Oklahoma City, OK 73112.

fore suggested that they result from impairment at two different embryonic stages.

Recently a molecular marker of certain anomalies such as congenital amputation was found. This marker is one of the homeobox-containing genes. These genes encode for proteins which bind DNA and regulate gene expression. It is likely that some cases of amputation are due to absence of the homeobox-containing or other genes.

Literature Review

Fromont[9] (1895) was the first to report an absence of median innervated thenar muscles. Su et al[27] reported a similar case, and Blair and Omer[3] reported a case of thumb hypoplasia and anomalous flexor pollicis longus (FPL) insertion. Tupper[28] reported four cases of hypoplastic thumb with anomalous insertion and course of the FPL tendon causing thumb abduction and loss of flexion and extension (pollex abductus).

Strauch and Spinner[25] reported eight patients with absent thenar muscles and FPL. Manske[16] treated fifteen patients with hypoplastic thumb and found that laxity of the MP ulnar collateral ligament is a consistent finding with aplasia of thenar muscles. Neviaser[22] reported eight patients with instability of the MP joint, adduction contracture of web space, and absent thenar muscles, extensors, and abductor pollicis longus. Ogino et al[23] reported ten cases of thumb hypoplasia and Nakamura et al[21] reported a family with thumb hypoplasia.

Dellon and Rayan[8] reported two cases of hypoplastic thumb with absent thenar muscles and without extrinsic musculotendinous anomalies, but the ulnar innervated thenar muscle FPB was present. Rayan[24] reported four patients with hypoplastic thumb in whom a single midline neurovascular bundle was noted, and a variety of extrinsic tendon abnormalities; all patients had spinal abnormalities.

Classification

Barsky[1] classified thumb deficiencies as hypodactylia or ectrodactylia. Blauth[4] classified thumb hypoplasia into five types of increasing severity. Type I is minimal hypoplasia or a short thumb due to decrease in size of osseous structures. Neurovascular and musculoskeletal structures are present and thumb function is normal. Type I is significant because of its possible association with several syndromes. Type II is moderate hypoplasia with thumb adduction deformity, MP joint instability and hypoplastic median innervated thenar muscles. Type III is severe hypoplasia with musculotendinous deficiency affecting both intrinsic and extrinsic components. The median and ulnar innervated thenar muscles are deficient or absent with absent or aberrant extrinsic tendons; abnormal tendon insertion and interconnections are possible. Type III may have intact metacarpal base and stable CMC joint (IIIA) or absent metacarpal base and unstable CMC joint (IIIB).[17] Type IV is subtotal aplasia (pouce-flottant) or floating thumb without function. It has no musculotendinous attachment or articulation with the hand, and the skin and neurovascular structures are the only attachment to the hand. Type V is thumb aplasia or absence of the thumb.

Hypoplastic thumb may be an isolated condition or associated with local differences or syndromes. Associated local abnormalities include syndactyly, constriction band (in utero amputation), polydactyly, radial club hand, cleft hand, arthrogryposis multiplex congenita, partial aplasia (congenital amputation and symbrachydactyly). In the last two conditions, similar aplasia of the fingers is frequently encountered.

More than twenty some syndromes may be associated with thumb hypoplasia. Short hypoplastic thumb may also be present in dwarfism conditions such as mucopoly-saccharidosis, achondroplasia, etc.

Clinical Picture

Thumb hypoplasia patients must be evaluated for general systemic and distant musculoskeletal abnormalities. Spinal abnormalities are particularly common[24] and may include such conditions as kyphosis and scoliosis with or without vertebral abnormalities, Sprengel's deformity, torticollis, and sacral agencies. Ocular, auricular, facial, cranial, and speech disorders may be present.

Thumb hypoplasia is a complex of several local abnormalities. The following areas should be examined carefully in a systemic fashion:

Thumb Size.— Comparing the hypoplastic thumb to the normal contralateral side can reveal subtle discrepancy in length or diameter. In an unpublished study of normal radiographs, we found the length of the thumb metacarpal, proximal, and distal phalanges to be 60% greater than proximal, middle, and distal phalanges of the small finger. Personal observations also showed that, in a severely hypoplastic thumb, the length of the metacarpal, proximal, and distal phalanges is frequently equal to or smaller than the proximal, middle, and distal phalanges of the normal small finger.

Web Space.— Web space deficiency may be due to skin or muscle contracture. In a previous study we used the goniometer to measure the

Non-operative treatment includes therapy and splinting for muscle contracture and functional activities to encourage the development of normal patterns of pinch and grasp.

Figure 1a. One-year-old child with typical cleft hand deformity. Both hands and feet have similar central defects. Additionally, patient has syndactyly of ring and small fingers and hypoplastic thumb with absent thenar muscles and shallow 1st web space.

Figure 1b. The patient's hand after reconstruction of the central cleft, release of ring and small finger syndactyly, reconstruction of the thumb hypoplasia, and local transposition flap to deepen the thumb index web space.

angle of thumb index web space outline.[12] We found the mean normal thumb-index web space angle to be 100°. Shallow or narrow web space can adversely affect hand functions such as picking up small objects or holding large objects. Web space contracture may be mild (60°), moderate (45°), or severe (30°). Absent web space is associated with syndactyly, as in Apert's syndrome. The web space angle and depth can be measured and compared to the normal contralateral side. Web space measurements can be used to evaluate postoperative improvement following web space reconstruction.

Intrinsic Muscles.— Absence of median innervated thenar muscles is frequently encountered with hypoplastic thumb. However, all intrinsic thumb muscles that are median and ulnar innervated should be evaluated, including the adductor pollicis muscle which may be absent or deficient, especially in Type III hypoplasia.

Extrinsic Muscles.— All extrinsic muscles of the thumb should be examined, including abductor pollicis longus (APL), extensor pollicis brevis (EPB), EPL, and FPL. Extrinsic musculotendinous units may be absent or have anomalous insertion. In absent FPL, loss of IP joint motion is associated with absence of IP joint crease.

Thumb Joints.— Each joint should be examined individually. The IP, MP, and CMC joints should be observed for active and passive ROM and for stability.

Thumb Function.— In infants this cannot be determined easily, but in older children the placing of objects in their hands and careful observation may reveal abnormal patterns of pinch and grip or lack of thumb opposition, flexion, or extension.

Treatment

Non-operative treatment includes therapy and splinting for muscle contracture and functional activities to encourage the development of normal patterns of pinch and grasp. Operative treatment of hypoplastic thumb should be tailored to the individual patient based on the findings and type of hypoplasia. Each deficiency should be given special attention and treated individually. A combination of surgical procedures is often necessary, and frequently several procedures can be done at the same time. The surgery is best done by the age of one year.

Size.—A short, otherwise normal thumb with functional extrinsic and intrinsic muscles requires no treatment. Metacarpal phalangization involves creating a thumb post by recession of intrinsic thumb muscles, including the adductor pollices and remnants of thenar muscles plus deepening of the web space. This procedure has functional limitations in congenital absence of the thumb because the intrinsic muscles are frequently deficient. Gradual distraction and lengthening using an external fixator can be used for short thumb and is done daily until desired length is achieved.

Web Space Deficiency.— If skin contracture is the cause, it can be treated by web space reconstruction using a variety of flap procedures such as the four flap Z plasty, transposition flap, or a fillet flap from the discarded remains of the polydactyly component or cleft hand (Fig. 1). If muscle contracture is the cause, it can be treated by releasing contracted muscles such as first dorsal interosseous muscle, adductor pollices, and anomalous musculus lumbricalis pollices.[14]

Thenar Muscle Deficiency.— Restoration of thumb opposition can be achieved by tendon transfer (opponenseplasty).[1] The motor muscle may be the abductor-digiti minimi (Huber transfer) or FDS.

Extrinsic Muscle Deficiency.— Restoration of extrinsic EPL or FPL function may require resection of the anomalous attachment, centralization of FPL,[28] rerouting,[3] or tendon transfer.

Joint Instability.— (a) Metacarpo-phalangeal joint instability can be treated by ligament reconstruction utilizing a slip from the superficialis[13]

or abductor digiti minimi[16] during opponeseplasty. Other techniques include capsulodesis, free tendon graft, chondroplasty by shaving the cartilage on both sides of the joint,[22] or MP joint arthrodesis, as in older patients. (b) CMC joint instability, if severe as in Type III hypoplasia, can be stabilized by bone graft, fusion to the adjacent index metacarpal, non-vascularized joint transfer, or vascularized metatarso-phalangeal joint transfer.

Floating Thumb.— Sparing the thumb remnants and reconstruction using metatarso-phalangeal joint transposition or free vascularized joint transfer can also be done. The functional results of thumb-sparing procedures are limited. Ablation and pollicization is the preferred method for treating "pouce flottant." The skin envelope of the hypoplastic thumb can be used for web space reconstruction.

Absent Thumb.— An abduction-pronation technique[11] by simple shortening of the index metacarpal, abduction, and rotation, does not give satisfactory cosmetic results. Toe-to-thumb microvascular tissue transfer[7,10,13,18] can be done, but the unpredictable anatomy of the neurovascular and musculotendinous structures of the thumb makes microvascular tissue transfer more difficult than in traumatic cases. This is especially true when aplasia is proximal to the MP joint and all intrinsic muscles are lacking. Functional recovery is limited by lack of full motion, soft tissue contracture, sensory recovery, and delay in cortical reeducation.[7]

Pollicization is the procedure of making a thumb out of an index finger. It was originally described by Buck-Gramcko.[5] Skin flaps are utilized to reconstruct a web space, the osseous structures are shortened and, additionally, rotation, fixation, and tendon transfers are done (Fig. 2). This is a complex and highly technical operation, but it is the reconstructive procedure of choice for the absent or severely deficient thumb. If the procedure is done properly, satisfactory functional and cosmetic results can be achieved.

Summary

Thumb hypoplasia is a common congenital deformity. Deficiency of intrinsic and extrinsic thumb muscles, web space deficiency, and joint instability can seriously compromise hand function. Clinical evaluation of patients with hypoplasia of the thumb should be conducted in a systemic fashion in order to accurately assess the extent of tissue involvement. The treatment goal should be improving hand function by addressing each area of deficiency individually.

Figure 2a. Type 4 hypoplastic floating thumb, with absent musculotendinous and osseous elements. This is a physiologically amputated thumb with no function and is equivalent to congenital absence of the thumb.

Figure 2b. The hand after ablation of the floating thumb and pollicization of the index finger. The patient's hand function improved and he was able for the first time to grasp, pinch, and oppose the new thumb to the adjacent middle finger.

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The Author

Ghazi M. Rayan, MD, is clinical professor of orthopedic surgery at the University of Oklahoma Health Sciences Center and also has a private practice in orthopedics, specializing in upper extremity, hand and microsurgery.

Lithotripsy for Retained Common Duct Stones: A Quasi-Urological Procedure

Donald D. Albers, MD; James L. Stammer, MD; James R. Wendelken, MD; Philip Mosca, PhD, MD

A protocol for bile duct lithotripsy was formulated in early 1988 after review of the literature and encouragement by gastroenterologists. Seven cases have been treated at our center and are being reported to explain our current concept of this therapy. Failure of the Food and Drug Administration to approve gallstone lithotripsy has complicated the promulgation of this procedure.

Retained common duct stones following cholecystectomy, occur in two to five percent[1] of patients. Subsequent endoscopic sphincterotomy with stone basket or balloon extraction is successful in 85% to 95% of these patients, however a few may require additional surgical procedures. Despite these additional procedures, a few retained stones may persist. A second surgical procedure may be considered too dangerous in certain high risk patients. In isolated situations, lithotripsy is an option, however it has not been approved by the FDA.

Lithotripsy (ESL) has been reported to be successful in the management of retained common duct stones.[2] Shock wave treatment (ESL) may only cause stone disintegration and thus be an aid to endoscopic extraction. In some cases, after ESL, there will be spontaneous passage of fragments and resolution of the problem.

Gallstone lithotripsy has been used extensively in Germany and a preliminary study in the U.S. has been reported in the *New England Journal of Medicine.*[3]

Direct correspondence to Donald D. Albers, MD, Oklahoma Lithotripsy Center, 5501 North Portland, Oklahoma City, OK 73112-2097

Lithotripsy in the United States is considered a purely urological procedure. Gallstone lithotripsy has not developed, and this is thought to be secondary to lack of FDA approval. A urologist localizes the stone and directs the lithotripsy in conjunction with the gastroenterologist. We have treated seven cases with retained common duct stones, and herein report our experience.

Materials and Methods

Six females and one male ranging in age from 44 to 92, with a mean age of 65 were treated. All patients had experienced previous procedures, including endoscopic sphincterotomy and attempted T-tube extraction, and, usually, surgical exploration. All patients had either a T-tube or an endobiliary tube in the common duct. All these unusual cases had large residual stones.

They were treated in the unmodified HM3 Dornier lithotripter, and the common duct stone or stones were visualized by injecting contrast medium through the T-tube or the endobiliary tube. The stone cast a negative shadow surrounded by contrast (Fig. 1). The number of shock waves (ESL) and kilovolt power (KV) and other details are summarized in Table 1.

Case Histories

Case 1.— A 65-year-old female had a cholecystectomy in 1987 followed by jaundice. Exploratory surgery was performed on 10/17/87 with removal of common duct stones and placement of a T-tube. Follow-up studies revealed more common duct stones. On 3/03/89 she had endoscopic retrograde cholangiopancreotography

Figure 1. Case 3—Showing large gallstone casting a negative shadow.

Figure 2 *(far right)*. Case 3—Common duct free of stone

(ERCP) and sphincterotomy. Balloon extraction was unsuccessful. A stone in the common duct measuring 2 ∞ 2.5 cm. was noted. Lithotripsy was performed on 3/31/88 without success. Additional surgery was necessary, and lithotripsy was considered a failure.

Case 2.— An 83-year-old man had a cholecystectomy in early May, 1989, and was later found to have a common duct stone. Surgical exploration was not successful in removing the stone. A T-tube was placed at the time of exploration. Treatment with ursodiol was carried out. Lithotripsy was performed twice. The stone decreased from 2 cm to 1.3 cm after the first treatment. A second lithotripsy was performed fracturing the stone to smaller fragments. A basket removal of residual fragments was performed. The patient is now stone free. Repeat open surgery was avoided.

Case 3.— A 53-year-old female had a cholecystectomy in January, 1987, and was later found to have a common duct stone. She had exploration and placement of a T-tube in the common duct. X-rays revealed a retained stone 1.6 ∞ 1.6 cm in size (Fig. 1). Lithotripsy was performed on 3/28/89. The stone was completely shattered. The T-tube was removed on 4/31/89. She is stone free (Fig. 2).

Case 4.—A 44-year-old female had a cholecystectomy in 1986 and had retained common duct stones. Three surgeries in a three-year period to remove stones from the common duct were performed. A 1.0 ∞ 1.4 cm stone remained. She had endoscopic retrograde cholangio-pancreatography and sphincterotomy and then lithotripsy on 7/31/89. The stone fragmented completely and by 10/02/89 all fragments were passed.

Case 5.—A 47-year-old female was found to have a 1.4 cm common duct stone following cholecystectomy in early 1990. She had exploratory surgery to remove the retained stone without success. She had ERCP and then lithotripsy on 5/30/90. The stone shattered and follow-up x-rays revealed no residual stones in the common duct.

Case 6.—A 92-year-old female was found to have a 1.2 cm stone in the common duct following cholecystectomy in mid-1992. She had ERCP

Table 1. Results of Bile Duct Lithotripsy in 7 Cases								
Patient No.	Age/Sex	Previous Exploratory Surgery	T-tube or Naso-biliary Tube	Stone Size	KV's	No. of Shocks	Success	Failure
1	65/F	x	x	2.0 cm x 2.5 cm	18	1650		x
2	83/M	x	x	2.0 cm	22	2400	x	
3	53/F	x	x	1.6 cm x 1.6 cm	22	2400	x	
4	44/F	x	x	1.0 cm x 1.4 cm	24	1500	x	
5	47/F	x	x	1.4 cm	18	2100	x	
6	92/F		x	1.2 cm	18	2000	x	
7	73/F	x	x	2.0 cm	18	2000	x	

with sphinterotomy and attempted stone removal without success. Lithotripsy was carried out on 10/7/92. The stone shattered successfully and the common duct is now free of stones.

Case 7.— A 73-year-old female had a cholecystectomy and was found to have a 2 cm and a 1.5 cm stone (Fig. 3) in the common duct. She had ERCP with attempt at removal but the attempt was unsuccessful. She then had exploration without success and a T-tube was placed. Lithotripsy was performed on 1/27/94. The stones were reduced considerably in size (Fig. 4). She was treated with ursodiol and was found to be stone free by x-ray (Fig. 5), and the T-tube was removed. She has had no further surgery.

Discussion

Shock wave lithotripsy for urinary calculi has been used since 1980 in Germany, and has become routine in the management of urinary calculi in the U.S. since 1986. In these cases the authors used a Dornier HM3, which uses spark plug generation of the shock wave with the patient in water. The shock wave is synchronized with a refractory period during the R wave of the electrocardiogram. The shock wave is generated by the firing of the spark plug at 18-24 kilovolts.

In these case presentations, success was limited, such as in patient No. 7. However, in all cases

Figure 3 *(far left).* **Case 7— Showing two negative shadows (gallstones) in the common duct.**

Figure 4. Case 7— Showing residual smaller stones in common duct.

Figure 5. Case 7—Common duct free of stones.

except patient #1, the stones were reduced in size with the use of lithotripsy. Treatment with ursodiol also helped reduce stone size. It is felt that lithotripsy may have saved additional open surgery. With urinary calculi, ancillary procedures are often necessary, such as endoscopic stone extraction. However, if open surgery is avoided, the lithotripsy is considered successful. Thus, lithotripsy was considered successful in six of the seven cases presented. These cases were performed through an experimental protocol with the Oklahoma Kidney Stone Research Foundation and the Deaconess Hospital Institutional Review Board (IRB).

In this country, the onset of laparoscopic cholecystectomy may have influenced the utilization of gallstone lithotripsy.

Several issues diminish the use of lithotripsy for treatment of common duct stones. Without FDA approval, the procedure provides no reimbursement for use of the lithotripter or for the physicians' time. It requires a urologist to donate his or her time, and coordinate scheduling with the gastroenterologists. At this time, the Food and Drug Administration (FDA) has not approved gallstone lithotripsy, and gives no indication of doing so in the near future. Despite lithotripsy's success in these case presentations, logistic difficulties and lack of reimbursement limits this treatment alternative for retained common duct stones.

Acknowledgments

Special thanks to John C. Carey, MD, for assistance with x-rays and to Marie Schoelen, RN, CNA, for redaction.

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The Authors

Donald D. Albers, MD, is emeritus clinical professor, Department of Urology, Oklahoma Health Sciences Center (OUHSC), Oklahoma City.

James L. Stammer, MD, is an Oklahoma City gastroenterologist.

James R. Wendelken, MD, is clinical associate professor, Department of Urology, OUHSC.

Philip Mosca, PhD, MD, is clinical associate professor, Department of Urology, and adjunct clinical assistant professor, Department of Pediatrics, OUHSC. He also has a private urology practice.

Progress Toward *Healthy People 2000:* A Preliminary Look at Oklahoma

Edward N. Brandt, Jr., MD, PhD; Robert W. Broyles, PhD; David J. Falcone, PhD; Neil E. Hann, MPH

Over 10,000 Oklahomans die each year from coronary artery disease or stroke. This study examined the behavioral risk factors for these illnesses present in Oklahomans. Oklahomans are at considerably higher risk than the desirable national goals for such risk factors. For example, too many Oklahomans smoke and too few exercise. The data to support these findings are included herein, and some steps to reverse these trends are recommended.

Strokes and coronary artery disease kill more than 10,000 Oklahomans each year, according to death certificate data. In addition, many more suffer nonfatal strokes and myocardial infarctions annually. Deaths attributable to heart disease and stroke are usually associated with behavioral risks such as smoking cigarettes, obesity and a sedentary life style.[1] Mortality from coronary artery disease and stroke is also associated with elevated levels of blood cholesterol and high blood pressure.[1]

The number of deaths resulting from heart disease and stroke is greatest among adults between the ages of 55 and 65, a group that might lower mortality rates by altering lifestyles. Unfortunately, many are exposed to risk factors that contribute to the onset of heart disease or a stroke. For example, approximately 33% of American adults are overweight, a problem that affects 35% of women and 32% of men.[2] Data assembled in the National Health and Nutrition Examination Survey reveal that 37% of women below the poverty level are overweight, an outcome that suggests obesity is associated with socioeconomic status. The results also indicate that the prevalence of obesity is disturbingly high among black and Hispanic people.

The control of weight and high blood pressure is contingent, in part, on exercise and diet. As of 1985, however, only 27% of obese Americans engaged in physical exercise and observed sound dietary practices to control their weight.[1] Further, less than 50% of adult Americans exercise regularly despite findings that suggest a sedentary lifestyle is an independent risk factor for coronary heart disease.[1] Of particular concern are findings that suggest older adults are less likely to engage in physical activity than their younger counterparts, thus enhancing the effects of aging.[3,4]

That cigarette smoking increases the risk of coronary artery disease and stroke has been well documented. Although the prevalence of cigarette smoking among Americans declined from over 40% in 1965 to less than 30% in 1987, smoking is associated with one in six deaths in the United States. Evidence indicates that more men than women smoke and that the rate of smoking is higher among black, Hispanic, and poorly educated people.

The effective design of policies to reduce illness or promote health requires information concerning the percentage of Americans who are exposed to health risks or engage in preventive

Deaths attributable to heart disease and stroke are usually associated with behavioral risks such as smoking cigarettes, obesity and a sedentary life style.

Direct correspondence to Edward N. Brandt, Jr., MD, Center for Health Policy, PO Box 26901, Oklahoma City, OK 73190.

practices. The purpose of this study is to examine the percentage of adults in Oklahoma who use preventive services or engage in activities that are believed to lower the risk of coronary artery disease or stroke. The use of preventive health services is represented by the percentage of Oklahomans who: (a) use prescribed medication to control their blood pressure, and (b) report a recent measurement of their blood cholesterol. The prevalence of obesity and the proportion of Oklahomans who smoke or engage in a sedentary life style serve as indicators for the broader set of behavioral risk factors that are associated with coronary artery disease or stroke. The data herein are for the period 1988-92.

Data and Methods

Healthy People 2000 has established a set of numerical objectives organized in 22 categories for improving the health of Americans to be achieved by the year 2000. These objectives were defined by a large group of physicians and public health personnel led by the U.S. Public Health Service. The objectives provide standards by which progress can be measured and, as such, represent a major contribution to our efforts to prevent disease.

The Oklahoma Behavioral Risk Factor Survey is administered annually to a statewide random sample of Oklahomans aged 18 years and older. Each question about risk factors was answered by the individual member of the sample and no attempt was made to verify their responses.

In this study, the objectives and goals established in Healthy People 2000 serve as standards for assessing changes in the prevalence of lifestyle risk factors and the use of preventive services by Oklahomans, while Oklahoma data were obtained from the five annual Behavior Risk Factor Surveys. The evaluation of changes in lifestyle risks and the consumption of preventive services is viewed from two perspectives. The first is based on pooled data from the five years that

Table 1. The Percentage of Adults in Oklahoma Who Had Their Blood Cholesterol Measured During the Preceding Five Years and, Among Those with High Blood Pressure, the Percentage Who Take Medication to Control Their High Blood Pressure, 1988-1992

Category		Year 1988	1989	1990	1991	1992	Pooled Percent	Year 2000 Goal
Measurement of Cholesterol	%	52.1	57.6	65.3	66.3	69.9	63.2	75 or more
	No.	967	1140	1341	1470	1467	6385	
Control of Pressure	%	62.1	64.3	61.8	53.7	57.5	59.2	80 or more
	No.	235	277	293	390	367	1562	

Table 2. The Prevalence of Obesity Among Adults in Oklahoma, 1988-1992

Category		Year 1988	1989	1990	1991	1992	Pooled Percent	Year 2000 Goal
Males	%	15.8	16.4	20.0	20.8	22.3	19.4	20 or less
	No.	354	440	556	582	547	2479	
Females	%	15.4	17.6	20.5	22.4	23.4	20.4	20 or less
	No.	577	660	743	831	870	3681	
Poor Females	%	18.5	28.0	32.4	23.3	30.3	26.4	25 or less
	No.	135	125	148	159	122	689	
Black Females	%	18.9	30.6	41.5	32.4	41.9	33.8	30 or less
	No.	37	36	53	68	43	237	

Frequencies and percentages based on computed values of the Body Mass Index

reflect the behavior of all respondents during the study period and compares the use of preventive care or the prevalence of behavioral risk with the goals established in Healthy People 2000. The second perspective focuses on the movement toward or away from established goals over the five-year period.

Results

The figures presented in Table 1 reflect the percentage of adults, 20 years of age and older, who reported that their blood cholesterol had been measured during the preceding five years. The pooled percentage, based on the responses of 6,160 adults, indicates that only 63.2% of the respondents had their blood cholesterol measured within a span of five years. Although the pooled percentage is less than the goal of 75% or more, the upward trend over the five years indicates that Oklahoma is moving toward established goals.

The data in Table 1 also suggest that only 59% of the 1,562 respondents with high blood pressure (which represents 25% of the total sample) rely on prescribed medication to control their hypertension. This figure compares unfavorably with the Healthy People 2000 goal of at least 80%. Further, the time series data exhibit a slight downward trend. These findings indicate that action is required to prevent an increasing number of the complications associated with hypertension.

The data presented in Table 2 depict the prevalence of obesity in Oklahoma during the study period. The results indicate that the goal of reducing the prevalence of obesity among adults to 20% or less was met in 1990 for both males and females. However, among poor and black females, the prevalence of obesity exceeded the established objective. Of particular concern are the apparent upward trends in the time series.

The findings listed in Table 3 suggest that the percentage of Oklahomans who engage in no physical activity, particularly among the poor and elderly, exceeds the recommended rate. These results also suggest that each of the time series exhibited an upward trend during the study period. Even though the data presented in this study

Table 3. The Percentage of Oklahomans Who Engage in No Physical Activity, 1988-1992

Category		Year 1988	1989	1990	1991	1992	Pooled Percent	Year 2000 Goal
18 or over	%	32.2	41.9	41.6	37.8	34.2	37.7	15 or
	No.	929	1100	1209	1413	1417	6158	less
65 or over	%	43.2	53.7	48.6	50.9	44.8	48.4	27 or
	No.	183	246	257	279	317	1282	less
Poor Adults	%	41.1	48.6	52.3	44.8	37.0	45.0	17 or
	No.	397	457	488	522	499	2313	less

Table 4. The Prevalence of Smoking Cigarettes Among Adults in Oklahoma, 1988-1992

Category		Year 1988	1989	1990	1991	1992	Pooled Percent	Year 2000 Goal
All Adults	%	25.4	28.1	26.5	27.1	26.6	26.8	15 or
	No.	931	1100	1299	1413	1417	6160	less
Blacks	%	29.8	25.5	26.8	24.2	25.8	26.2	18 or
	No.	57	55	82	95	66	355	less
Education (High School or Less)	%	29.8	32.0	31.1	33.1	29.3	31.1	20 or
	No.	476	571	626	712	706	309	less
Reproductive Females	%	25.8	29.6	29.0	27.5	28.1	28.0	12 or
	No.	287	311	365	407	420	1790	less

It is critical that we begin to address the issue of reducing the risks of Oklahomans for heart disease and stroke.

fail to reflect physical exercise that accompanies work-related activity, the results hardly bode well for the future progress toward the Healthy People 2000 goal.

When compared to the goal of reducing the prevalence of smoking to recommended rates, the results listed in Table 4 suggest that the percentage of Oklahomans who reported smoking cigarettes is excessive. With the exception of black adults, these findings also suggest that the percentage of Oklahomans who smoked cigarettes increased during the study period, an outcome that is perhaps most serious among females of reproductive age.

Discussion

This overview of health risk behavior is hardly encouraging. Undoubtedly, Oklahoma's relatively poor performance is due, in part, to the low socioeconomic status and educational attainment of its residents. Traditional medical and public health efforts will have no effect on socioeconomic status or educational attainment and, therefore, will have little direct effect on the root of the problem. Having acknowledged that fact, the challenge to health promotion and education is formidable, but it also is irresistible and immediate. The limited data presented here are a part of a stark baseline against which the results of taking on the challenge can be measured.

What Can Be Done?

It is critical that we begin to address the issue of reducing the risks of Oklahomans for heart disease and stroke. There are a number of steps that physicians can take to address these risks.

- Physicians can be alert to the risk factors present in their patients and help them reduce their health risks by education and other appropriate means.
- Physicians can cooperate with the state department of health and local health departments to educate Oklahomans as to their risks.
- Physicians should insist that their legislators support legislation that would include such things as harsh penalties for persons selling cigarettes to people under 18, and coverage for preventive services by insurance plans especially by state-sponsored programs such as Medicaid and the state employees health benefit programs.
- Physicians can set an example by practicing prevention and engaging in the early detection of disease.

None of these steps alone is a cure but, as a whole, they can make significant progress to reduce disease, suffering, and death, which, after all, is the mission of medicine.

The Authors

Dr. Brandt is professor, Departments of Health Administration and Policy (DHAP) and Medicine and director, Center for Health Policy Research and Development (CHPRD), University of Oklahoma Health Sciences Center (OUHSC), in Oklahoma City.

Dr. Broyles is professor, DHAP, and member, CHPRD, OUHSC.

Dr. Falcone is professor and chairman, DHAP, and member, CHPRD, OUHSC.

Mr. Hann is director, Office of Tobacco Use Prevention, Oklahoma State Department of Health.

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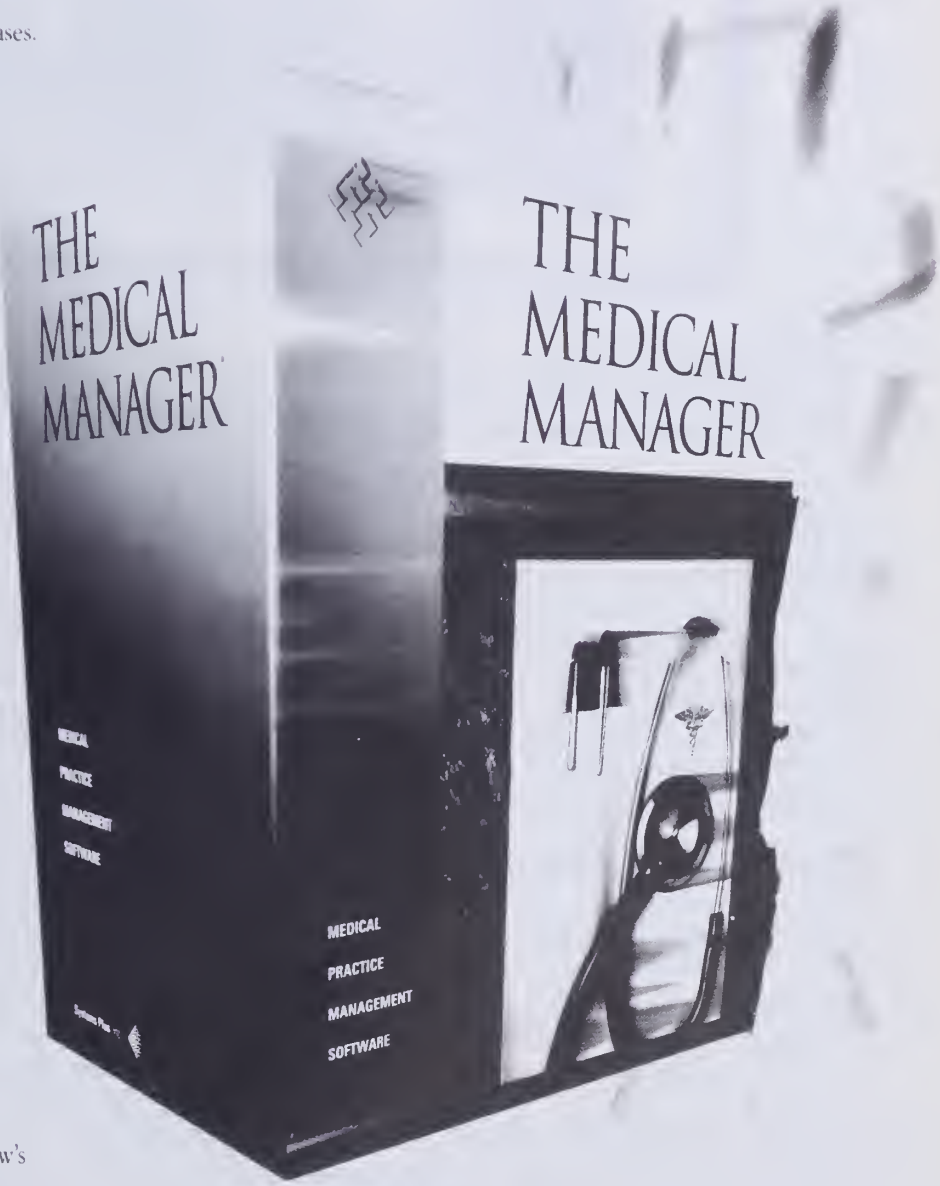

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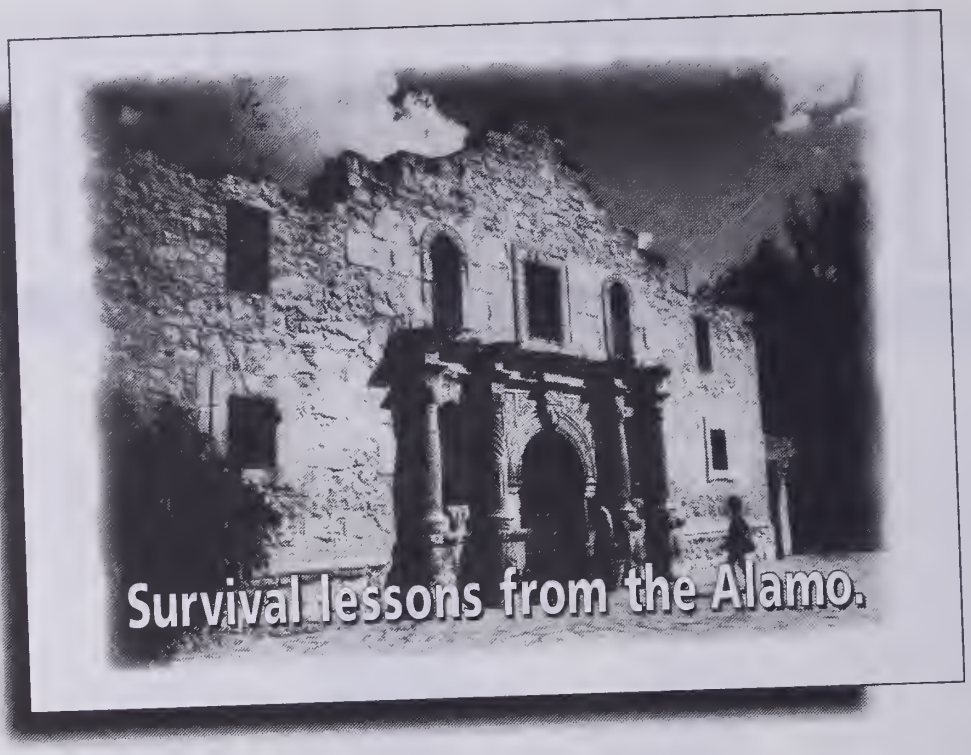

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HEALTH DEPARTMENT

Hemophilia now reportable for purposes of special state survey

The Oklahoma State Department of Health is one of six states nationwide participating in a three-year project funded by the Centers for Disease Control and Prevention (CDC) that will focus on better describing morbidity associated with the two most common hemophilias, Factor VIII and Factor IX deficiencies (hemophilia A and B, respectively). The purpose of the project is to determine the prevalence of hemophilia A and B in Oklahoma and to determine the frequency and types of complications related to the disease. The project's ultimate goal is to reduce the human suffering and health care costs associated with hemophilia. This project is being conducted in collaboration with the Oklahoma Hemophilia Treatment Center and the Department of Biostatistics and Epidemiology, OUHSC College of Public Health.

To determine prevalence, the Commissioner of Health has declared hemophilia a reportable disease for the purposes of this special investigation (Oklahoma Administrative Code 310:515-1-6 and 63 O.S., Section 1-106). This allows project staff to ascertain cases of hemophilia. Once identified, project staff will conduct confidential medical record reviews. Based on the incidence of hemophilia and the population of Oklahoma, the estimated prevalence is 315 cases. Sources of potential cases include the Oklahoma Hemophilia Treatment Center, hospital discharge summaries, selected medical providers, IHS facilities, and commercial laboratories. Once the cases are identified, data abstractors will review medical records. Information related to complications will be abstracted and submitted to the CDC, without patient identifiers, for further analysis. Medical records will be reviewed annually to determine the occurrence of new complications in previously identified cases and to ascertain new cases.

The project, which is funded from September 1993 through September 1996, has begun the process of case ascertainment. If you care for or know of patients with either hemophilia A or hemophilia B, please contact the project coordinator, Barbara Erickson, at 405-271-3970 (collect calls accepted).

Hemophilia Overview.— Two of the most common hemophilias Factor VIII deficiency (hemophilia A) and Factor IX deficiency (hemophilia B), are sex-linked single gene disorders that affect an estimated 1 in 5,000 males, reports the Oklahoma State Department of Health. Affected males experience both spontaneous internal bleeding and bleeding following injuries or surgical procedures. Repeated bleeding episodes produce numerous associated complications, such as severe joint damage, stroke, or other neurologic damage, and damage to other organ systems that are affected by hemorrhage. With the introduction of clotting factor concentrates in the late 1970s and early 1980s, individual bleeding episodes were able to be treated more quickly and efficiently. Although factor replacement therapy improved the management of hemophilia, it also introduced other complications. Prior to heat treatment of factor concentrate in 1985, affected individuals were at an increased risk of contracting hepatitis and HIV. Some estimates indicate that as many as 50% of those affected developed chronic liver disease or AIDS from factor concentrate use. While the etiology and pathogenesis of Factor VIII and Factor IX deficiency is well understood, little is known about the frequency or natural history of complications that result from the disease and its treatment.

Programs for persons living with HIV disease

The Oklahoma State Department of Health (OSDH), with the assistance of Ryan White Title II funds, is administering programs designed to assist low income individuals who are living with HIV disease. These programs are the HIV Drug Reimbursement Program (HDRP) and the HIV Home Health Program (HHHP). HDRP is designed to provide certain specified drugs related to HIV disease. Recipients must have a documented HIV infection, meet certain eligibility requirements, and have been prescribed treatments which have been determined to prolong life or prevent the serious deterioration of health arising from HIV disease.

Medications on the HDRP formulary are as follows: pentamidine isethionate (Nebupent), didanosine (ddI, Videx), zidovudine (AZT, Retrovir), fluconazole (Diflucan), trimethoprim and sulfamethoxazole (Bactrim, Septra), zalcitabine (ddC, Hivid), and mycobutin (Rifabutin).

(continued)

HEALTH DEPARTMENT (CONTINUED)

The HHHP is designed to assist low-income individuals who are living with HIV disease with in-home care as an alternative to repeated or prolonged stays in hospitals or nursing facilities, thereby enhancing the quality of life while reducing treatments costs. The HHHP will cover personal care visits, skilled care visits, and durable medical equipment and/or supplies.

In addition to these programs, Health Maintenance Clinics in Lawton, Enid and Ardmore are scheduled to re-open in April. The purpose of these clinics is to provide primary intervention to individuals living with HIV disease in parts of rural Oklahoma.

Additional information can be obtained from the Oklahoma State Department of Health at 405/271-4636. For information regarding HDRP and Health Maintenance Clinics, contact Robn Muldowney. For information regarding HHHP, contact Mike Gilliam.

Neural tube defects and folic acid supplements

Between April 1992 and August 1993, in a rural Oklahoma county, five children were born with a neural tube defect (NTD), specifically myelomeningocele. While the expected rate for myelomeningocele ranges between 3.1 and 7.8 per 10,000 live births, this county had a rate greater than 1 per 400 live births in 1992 and 1993. Although the cause of NTDs is unknown, a method of reducing the incidence of NTDs in Oklahoma is available.

It is recommended that women with a prior NTD-affected pregnancy receive 4.0 mg (400 g) of folic acid per day one month prior to conception and throughout the first trimester to reduce the risk of NTD recurrence.

This recommendation was made by the Centers for Disease Control and Prevention (CDC) in 1991. Since 1976, it has been recognized that there is a relationship between deficient folic acid intake and neural tube defects. Studies conducted by the British Medical Research Council demonstrated that the use of high-dose folic acid supplements by women with previous NTD-affected pregnancies reduced the risk of subsequent NTD-affected pregnancies by 70%. This dose should only be taken under a doctor's supervision since it can complicate the diagnosis of vitamin B_{12} deficiency.

In 1992, it was recommended that all women of childbearing age capable of becoming pregnant consume 0.4 mg (400 g) of folic acid daily to reduce the risk of having a pregnancy affected with spina bifida or other NTDs. This recommendation includes women with a pri-

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or NTD-affected pregnancy unless they are planning a pregnancy.

Recent studies indicate that periconception folic acid supplementation reduces the incidence of the first occurrence of NTDs by as much as 60%. This Public Health Service (PHS) recommendation can be accomplished by taking a multivitamin containing 0.4 mg (400 g) of folic acid or by eating a diet high in folic acid. After conception, a prenatal vitamin should be taken daily unless there has been a previous NTD occurrence. Since the effects of high intake of folic acid are not well known, consumption of folic acid should be limited to <1 mg daily except under the supervision of a physician. The CDC estimates that following the PHS recommendations will avert 50% of NTD cases.

The risk of an NTD-affected pregnancy is greater in women who have had a previous NTD-affected pregnancy, yet 95% of affected pregnancies occur in women with a negative history. Therefore it is important for all health care providers to discuss folic acid intake through diet or vitamin supplementation as a means of reducing the risk in all women capable of childbearing. Risk reduction will be limited if only women who are planning pregnancies are targeted, since approximately 45% of Oklahoma births are the result of unintended pregnancy. This recommendation does not eliminate the need to offer maternal serum alpha fetoprotein (MSAFP) screening during the prenatal period.

For more information about folic acid supplementation, contact the Oklahoma State Department of Health, Maternal and Infant Health Service, Congenital Disorders Division at (405) 271-6617.

WORTH REPEATING

Doctor still practicing at 80; has no plans for retirement

BY LAURIE WINSLOW
TULSA WORLD STAFF WRITER

People frequently ask Dr. Rayburne Goen when he plans to retire. At age 80, he has no plans whatsoever.

"I will continue until I'm old enough to be put out to pasture," Goen said, while seated behind his office desk. "I love what I'm doing. I'd rather do this than anything I can think of."

Goen specializes in internal medicine and cardiology with the Wheeling Medical Group, 1919 S. Wheeling Ave.

The doctor has been practicing medicine for nearly 55 years. On Thursday, his office staff helped him celebrate his 80th birthday.

WORLD STAFF PHOTO BY MIKE M. RUIZ

Dr. Rayburne Goen

Hard work has never been known for killing anybody, Goen said. He works as long today as he did when he first started, frequently putting in 12 to 13 hours a day. He takes night and weekend calls. Occasionally, he still makes house calls.

This old-fashioned courtesy is reminiscent of a past when doctors daily paid visits to their clients. Goen remembers with clarity the days of doctoring nearly 50 years ago.

Doctors didn't have antibiotics. They couldn't perform open heart surgery, he said. Their doctor's kit included necessities such as Epsom salts, castor oil, aspirin, iodine, mustard plaster but no Band-Aids.

"I never used leeches. I came along a little after that," he said with a smile. "Hospitals were small and crowded. They had difficulty getting staff."

Goen became interested in entering medicine as a high school student, living in Texas. His family's dentist encouraged him to become a doctor.

"It's interesting. I'm the first person on either side of my family who went past eighth grade," Goen said.

Goen studied pre-med one year at Texas Tech University in Lubbock before enrolling in the University of Colorado. He graduated from medical school in 1939.

He was within a couple of weeks of fulfilling his commitment at Fitzsimons Army Medical Center in Colorado when the attack on Pearl Harbor occurred. Goen dedicated four more years to the military.

Goen and his wife, Sally, moved here in December 1945 and in January he opened his first office.

A lot has changed from those early days when house calls cost $5 and office visits cost $3.

One thing that never has changed is Goen's love for his job.

"No. 1, I like people. I like the feeling that I'm able to help them or coun-

WORTH REPEATING (CONTINUED)

sel them," he said. "I'm not ambitious about fame or fortune, but satisfaction is high."

The lives and stories of long-time patients pack his memory.

Goen recalled the time one of his elderly patients, a 101-year-old woman, was having trouble breathing. An attendant at the nursing home where the woman was staying called Goen and told him the woman's skin was clammy and she couldn't lie down.

It was an icy November day, Goen said, and by the time he reached the patient's bedside, he explained that his hands and stethoscope were cold.

"She said, 'Doctor, you can do anything you want with me, but just don't mess up my hair.' Vive la difference," Goen said, chuckling.

Longtime patients speak highly of him.

"We are so fond of him," said Mary Ann Elliott, who has been going to Goen for the past 20 years. "He fools you. He gives the impression he's a country doctor, but he knows everything."

Betty McKenney, 73, said both she and her husband, Stan, have been going to the doctor for as long as they can remember. He treated both their parents.

"He made house calls. It didn't matter if it were day or night. He was so wonderful," she said.

Goen is now absorbed with the task of creating a computer system that responds to voice commands and compiles comprehensive data on patients.

Goen believes it is his love for his work that keeps him young at heart and mind. He does calisthenics every morning and spends a lot of time taking care of his garden and cabin at Lake Fort Gibson.

"I do most everything the hard way," said Goen, who never takes the elevator and parks his car at the farthest lot.

"My wife has been a wonderful help and inspiration to me."

Before they married, she accepted the fact he would be married to his job, Goen said.

The couple have four children, eight grandchildren and two great-grandchildren.

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LETTERS

Health care reform going underground

To the Editor: *And the bad news is:*

Healthcare reform is not dead. Hillary Clinton blames bad politics for the failure of a healthcare reform bill to make it to a vote before the recess. The latest announcement from the administration is that it will be back in 1995 but with the political error corrected: Mrs. Clinton and Ira Magaziner will remain in the background like puppeteers, pulling the strings—but you won't see them.

Mrs. Clinton added that people in this country had been trying for decades to get government -run, universally mandated healthcare through the Congress.

In her mania for power, Clinton does not recognize that since Wilbur Cohen first tried to socialize the entire provision of medical care in American in the mid-1930s, hundreds of millions of Americans have repeatedly fought to stave off the efforts of a few dozen members of the Congress, a few presidents and various other "liberals," both within and outside of the political structure, who actively seek a politically dominated, government controlled, semi-European style of socialized medicine. Who in his right mind wants it? One more time we Americans have managed to convince a majority of members of the 103rd Congress that we did not even trust a vote on the matter lest something get slipped off on us as has so often happened before.

With healthcare reform going underground, the need for citizen vigilance is greater than ever. Just as 3/4 of Medicare/Medicaid legislation has been slipped through as riders on other bills, we can expect to find next year's healthcare reform bills tacked piecemeal onto bills such as those concerning public lands, banking, wetlands conservation, etc.

We're going to get it, folks. We're just not going to see it coming unless we go after the 104th Congress right away.

—*George C. Manning, MD*
Fort Wayne, IN

Note: *Dr. Manning's letter was written on the eve of the November 8 elections.* —Ed.

Rural Health

18th Annual National Conference
National Rural Health Association
May 17–20, 1995
Atlanta, Georgia

OSMA directories available

The *1995 Oklahoma State Medical Association Directory of Physicians* is now in distribution. All OSMA members will receive a complimentary copy. Additional copies are available to members at $25 a copy and to non-members at $50 a copy. Bulk orders of ten or more directories may be purchased at $25 per directory. Make checks payable to the OSMA, 601 Northwest Expressway, Oklahoma City, OK 73118.

Directory corrections should be mailed to OSMA Membership Coordinator Judy Lake and will be published in an upcoming issue of the JOURNAL .

DEATHS

Clifford Leslie Lorentzen, MD
1922 - 1994

OSMA Life Member Clifford L. Lorentzen, MD, died September 9, 1994, in Ardmore. Dr. Lorentzen was born in Powers Lake, N.D., where he grew up. He was a graduate of the University of North Dakota and the University of Illinois Medical School in 1947 and served his residency in internal medicine and cardiology in Chicago. A World War II veteran, Dr. Lorentzen served in the U.S. Army. He also served as an Air Force physician during the Korean conflict. Active in organized medicine, he was a past president of the Oklahoma Society of Internal Medicine and of the Carter, Love, and Marshall County Medical Association. He also was medical director of the Carter County Heart Association for 20 years.

Roger James Reid, MD
1903 - 1994

Dr. Roger J. Reid, a native of Winnipeg, Manitoba, Canada, died September 26, 1994, at his home in Ardmore. He was graduated from the University of Manitoba in 1928 and the faculty of medicine in 1932. He served with the U.S. Army Medical Corps during both World War II and the Korean conflict, and was commander of the 179th and 120th Medical Battalion 45th Infantry. Dr. Reid was a past president of the Tri County Medical Association and the Ambulance Board, chairman of the Medical Examiner's Board, a delegate to the American Medical Association, and a charter member of the American Academy of Family Practice in Oklahoma. He was named an OSMA Life Member in 1977.

David George Schrouf, MD
1965 - 1994

Oklahoma City native David G. Schrouf, MD, died November 5, 1994, in his hometown. An ophthalmology resident, Dr. Schrouf earned his medical degree at the University of Oklahoma College of Medicine, where he was graduated with Special Distinction in 1991. He was also a member of Alpha Omega Alpha, an honor medical society representing the top 10% of medical students and professionals nationwide.

IN MEMORIAM

1993

Eric McLain White, MD	April 23
John Franklin DeJarnette, Jr., MD	May 1
Harold Alwin Smith, MD	May 4
Kenneth Warren Bonham, MD	May 28
John Raymond Stacy, MD	July 6
Carl Davis Osborn, MD	July 11
Mark Royal Johnson, MD	July 12
W. Turner Bynum, MD	July 13
James Dougherty Martin, MD	July 31
Edward David Greenberger, MD	August 6
Horace Harold Porter, MD	August 15
Clifford A. Traverse, MD	August 20
J. Wendall Mercer, MD	September 2
Emma Jean Anthis, MD	September 27
Russell D. Harris, MD	September 28
Walter Lee Honska, Jr., MD	October 9
Ethel Maurice Walker Heras, MD	October 25
James Burton Pitts, Jr., MD	October 29
John Daniel Capehart, MD	November 20
Kevin James Cammack, MD	December 4
William Best Thompson, MD	December 10
Henry Grady Ryan II, MD	December 17

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Kirk Thornton Mosley, MD	January 3
Richard Charles Wade, MD	January 6
Austin Walsh Webb Haddox, MD	January 13
Earl Mathiews Woodson, MD	February 20
Tom Lamar Johnson, MD	March 5
Orville Main Rippy, MD	March 11
Minor Elliott Gordon, MD	March 14
George Loren Norris, MD	March 27
Max A. Glaze, MD	April 29
Winfred Aaron Showman, MD	May 14
Mark Daniel Holcomb, MD	June 1
Carter William Mathews, MD	June 3
Frank Wilson Clark, MD	June 6
Harold Ray Sanders, MD	June 15
Robert Bruce Howard, MD	June 16
Richard Warren Loy, MD	July 7
John Hobson Veazey, MD	July 11
Wesley A. Whittlesey, MD	July 12
Laurence Sevier McAlister, MD	July 19
Jon Meyer Chenette, MD	August 4
Beryl Drew Henwood, MD	August 14
Jess Duval Herrmann, MD	August 16
Clifford Leslie Lorentzen, MD	September 9
Roger James Reid, MD	September 26
David George Schrouf, MD	November 5

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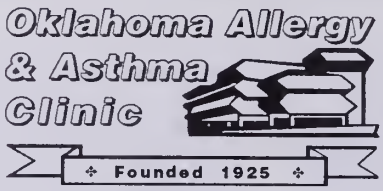

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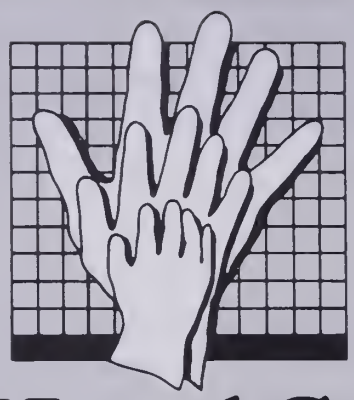

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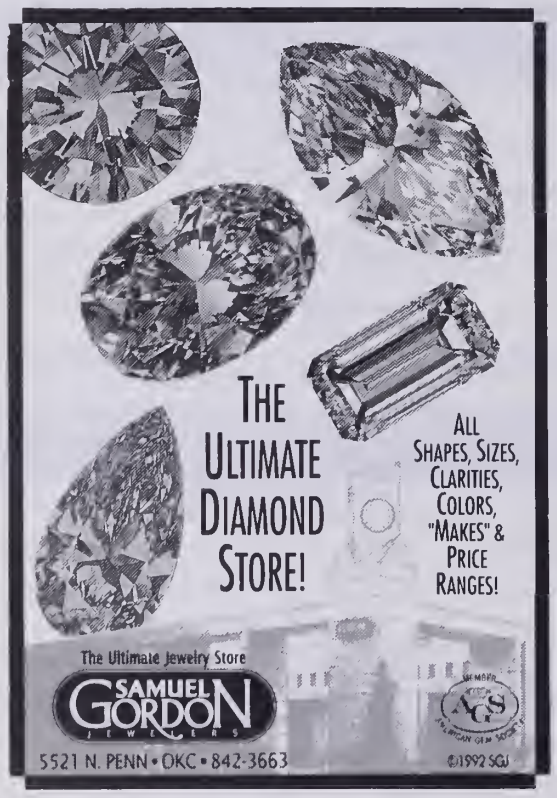

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■ **The 22nd Annual Critical Care Medicine Course to be** held March 4-9, 1995, at the Oklahoma City Marriott Hotel, is a 5-day intensive educational experience featuring didactic presentations and workshops. Accreditation of 48 hours is awarded by the AMA, AAFP, AOA, and ACEP.

The course is presented and sponsored by the Department of Medicine, College of Medicine, University of Oklahoma Health Sciences Center and the Veterans Affairs Medical Center in conjunction with the Office of Continuing Medical Education. Program co-directors are Paul V. Carlile, MD, and Martin H. Welch, MD. Tuition is $695 (house staff/allied health $525).

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Guest speakers include nationally known physicians Donald D. Trunkey, MD, from Oregon Health Sciences University; Alfred F. Connors, Jr., MD from Case Western Reserve University School of Medicine; Alan H. Morris, MD, from the University of Utah; and L. David Hillis, MD, from the University of Texas Southwestern Medical Center.

For further information please contact Dora Lee Smith, Course Coordinator, at (405) 271-5904 (Fax: 405-271-5892), or write to: Critical Care Medicine Course, University of Oklahoma Health Sciences Center, Room 3SP-400, P.O. Box 26901, Oklahoma City, OK 73190.

■ **February 1, 1995 is the deadline for nominations for** the Ayerst Wyeth Physician Award for Community Service and the Donald J. Blair Friend of Medicine Award. The service award goes to a physician who, in addition to his or her medical practice, has contributed significantly to the community. The Blair award, formerly the Layman of the Year Award, is presented to a non-physician for his or her support of the art and science of medicine in Oklahoma. Winners are selected by the OSMA Board of Trustees at their February meeting each year and the awards are presented at the Annual Meeting of the OSMA House of Delegates in April. Winners in 1994 were Bertha M. Levy, MD, Oklahoma City, and Wilson D. Steen, PhD, Norman.

Nominations for both awards should be sent with supporting material to Douglas C. Hubner, MD, Chairman, OSMA Board of Trustees, 601 Northwest Expressway, Oklahoma City, OK 73118.

■ **Tomas P. Owens, MD, Edmond, a faculty member in** the University of Oklahoma College of Medicine's Department of Family Medicine in Oklahoma City, has been elected to Alpha Omega Alpha, an honor medical society representing the top 10% of medical students and professionals nationwide. Although most members are elected during their junior or senior years of medical school, physicians who have demonstrated outstanding leadership and research success after graduation also are eligible for membership. Dr. Owens was nominated for the honor by the OU College of Medicine chapter. Other department members of AOA are Peggy L. DuBois, MD, Midwest City; Kenneth R. Smith, MD, Edmond; John P. Zubialde, MD, Norman; and James W. Mold, MD, Edmond.

■ **Vadakepat Ramgopal, MD, Oklahoma City, is a member** of the AMA's International Medical Graduate Advisory Committee.

■ **M. Joe Crosthwait, MD, Oklahoma City, has been** appointed to the AMA's Advisory Committee on Senior Physicians.

■ **The National Rural Health Association will hold its** 18th annual national conference on rural health, "Rural Health—Harnessing the Winds of Change," May 17-20, 1995, at the Hyatt Regency Atlanta Hotel in Atlanta, Ga. The conference will focus on helping rural health care providers and systems deal with the changing trends in rural health care brought about by the national health reform debate. Additionally, it will explore how to address these recent changes in health care to benefit providers and health care professionals and develop future rural health models of care and united strategies that will help attendees deal with the emerging health care environment.

The conference is designed to develop practical skills and techniques in health services administration, organization, and research; present practical sessions of interest to rural health providers; showcase current policy issues affecting rural health services; and report the results of research applicable to rural health service administrators and providers.

The education program of the conference is tailored to the needs of hospital and clinic administrators, physicians, nurses, researchers, policymakers, educators, and state and federal officials. It features more than 60 general and concurrent sessions, topical symposia and contributed research papers conducted by leaders in the rural health field. Each year the conference draws more than 1,000 participants. The conference program also includes exhibits, as well as national awards presentations recognizing outstanding achievement of rural health care providers, practices, programs, education and research. To receive registration information, contact the HRHA National Service Center, One West Armour Blvd., Suite 301, Kansas City, Mo., 64111, 1-816-756-3140.

■ **James R. Rhymer, MD, Clinton, has been elected to a** two-year term as vice-chair of the Oklahoma Merit Protection Commission.

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